THE JOURNAL OF PERIODONTOLOGY

vol. 9

January - July
1938

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Published by
THE AMERICAN ACADEMY OF PERIODONTOLOGY
VOLUME 9.
NUMBER 1.

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v. 9

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THE JOURNAL OF PERIODONTOLOGY is published to serve as the professional journal of periodontia; to keep dentists in touch with research being carried on in this field and related sciences; to stimulate greater interest in the supporting tissues of the teeth and their relation to oral and general health. The plan to continue the publication of abstracts indefinitely will make the Journal a fund of extensive information, readily available to students, teachers, researchers and clinicians.

The Journal is the official organ of the American Academy of Periodontology and will contain therefore those papers of scientific interest which are presented at the annual meetings of that society. The proceedings of the 1937 annual meeting are published in this number. The July, 1938 issue will contain the abstracts of articles on periodontia which appeared during the year of 1937.

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"It is eminently fair to expect all dentists to discover periodontal lesions, regardless of their disposition in respect to treatment. Though relatively few readily adapt themselves to the exacting demands of this phase of modern practice, the profession generally cannot escape the responsibility of at least competent examination, intelligent diagnosis, and reference of cases."

—Justin D. Towner (See p. 48.)

THE JOURNAL OF PERIODONTOLOGY

JANUARY, NINETEEN HUNDRED THIRTY-EIGHT

Gingival and Periodontal Disease In Children[1]

By JOHN OPPIE MCCALL, D.D.S., F.A.A.P., F.A.C.D[2]

WHEN John Riggs announced that suppurative periodontal disease was curable and demonstrated that, in his hands, the disease could at least be checked, he inaugurated the modern era of periodontology. Advance, after the demonstrations of Riggs, was slow and fitful. And only within the span covered by the history of this Academy, and, in fact, largely through the stimulus and help given by this society, have the science and the art of periodontology been established on a firm foundation. During the past twenty-five years, in which such important progress has been made, attention has centered almost exclusively on periodontal disease in the adult mouth. Even the study of periodontal etiology has been carried on chiefly in the adult mouth, although the relationship of malocclusions originating in childhood, to the causation of periodontal disease in later life has been given considerable attention.

With all that has been done in that field, however, and with all the dissemination of the findings of students of periodontal etiology through the literature, there is still a lack of full appreciation of the fact that the foundation of virtually all periodontal disease is laid in childhood and a corresponding lack of interest in the condition of the periodontal tissues in the mouths of children. Wordsworth wrote, "The child is father of the Man," and this phrase, so often quoted, has seldom had better application than in the case of the topic we are here considering.

But even if we were not concerned with the relationship of juvenile periodontitis to periodontoclasia in the adult mouth we ought now to turn to the consideration of gingivitis and deeper periodontal disturbances in the mouth of the child, as a distinct and important phase of periodontology.

I do not mean to imply that this field has been entirely neglected, for we have, of course, the very excellent research by Hanke[3], made at the Mooseheart Home, some years ago. I do feel, however, that further studies should be made in this branch of periodontal pathology, and I believe that these should be carried on by, or in close collaboration with, experienced periodontists. This paper

[1]Read before The American Academy of Periodontology, July 8, 1937.
[2]Director, The Murry and Leonie Guggenheim Dental Clinic, New York City.
[3]Diet and Dental Health. Milton T. Hanke, University of Chicago Press, 1933.

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is intended as a preliminary survey of this field and it is hoped that it will stimulate others to make more complete studies.

During the past six years I have had an unusual opportunity to observe a large number of children ranging in age from about two to fourteen, children who have been admitted to the Murry and Leonie Guggenheim Dental Clinic. At the outset we must consider certain facts that apply to these children as a group. Admissions are restricted to indigent children living in the immediate vicinity of the Clinic, New York's upper East Side. Sixty-five per cent of the children in the public schools of the district are considered eligible for admission and this figure will enable you to visualize to a degree the living conditions and health conditions that prevail. I do not wish to imply that conditions in this district or in other of the poorer parts of New York are altogether detrimental to health. This is far from being the case. Dietaries in most families are fairly varied and contain, in most cases, at least minimal amounts of vitamins and minerals. Fresh air and sunshine are found in greater amount than is commonly assumed. The children play on the sidewalks and streets, and in playgrounds that are being provided to an increasing extent. New York children lack to a degree one important health aid and that is adequate and properly timed rest. This is due to improper housing and the noises of a big city. Bad housing conditions exert an unfavorable influence in other ways, also. That health conditions are not entirely bad, however, is indicated by the fact that we see, among those coming to the Clinic, many beautiful children, the picture of health, with bright eyes, good color, and firm flesh. Yet even among these there are few who are free from some degree of dental caries and gingivitis. Since surveys made in other cities, even the smaller ones, indicate that dental conditions in them are similar, I dare to hold up the conditions found in New York as typical in kind, and virtually in extent, of those found elsewhere in urban communities.

Children are admitted to the Clinic, not because they have been found to need dental care, but solely on the basis of financial eligibility. We, therefore, get an accurate cross section of the child population. As far as having had previous dental care is concerned, they are virgin soil, and we, therefore, see what happens at the various ages when teeth are neglected. Dental caries is found in over ninety-eight per cent of these children. It may begin at the age of two, sometimes before that age; fifty per cent of the two-year-olds admitted to the Clinic have decaying teeth. Gingivitis is nearly as prevalent, but does not usually put in an appearance so early. At four or five years of age we begin to see characteristic gingival lesions in the mouths that have had untreated caries. From this point on we see very few mouths that do not show at least a mild gingivitis of a localized if not a generalized character.

The point now is not whether periodontal disease exists in childhood but what type or types are to be found and, more important, whether the disturbances of the supporting tissues found in childhood are the precursors of the

periodontal lesions so widely distributed in the adult population. If the latter proves to be the case, the question arises as to whether or not emphasis must be transposed to the earlier years of life in the preventive periodontal program.

Quite a wide variety of lesions of the gingivae and other periodontal tissues are found, and it is therefore necessary to propose a classification under which they may be organized for study. We may, of course, use the older classification of gingivitis, recession, alveoloclasia, pericementoclasia. These terms, while convenient as descriptions of clinical entities, suggest very little as to the essential pathological processes involved. It seems best, therefore, to turn to the classification proposed by Box, viz., *gingivitis, periodontitis simplex* and *periodontitis complex*. To this I think it well, however, to add *stomatitis,* meaning by this a generalized inflammation of the soft tissues of the mouth, with, usually, special involvement of the gums.

The manifestations of the various types and subtypes of disease under the above classification differ so much, in many instances, from those of the corresponding conditions in the adult, that there may seem, at first glance, little in common between them. Furthermore, pus pocket formation, so commonly considered to be *the* typical phase of periodontoclasia in the adult, is conspicuous by its rarity in the child. Relationships can be established, however, and results both in research and in treatment will be most productive, if we seek out such relationships in classification and etiology as do exist.

In Box's classification, gingivitis may occur alone or as a phase in either periodontitis simplex or complex. In the adult it is nearly always seen as a part of a case of periodontitis, but in the child we find it very often *solo,* that is, occurring without accompanying disease of either alveolar bone or pericementum. This is so common a finding that it is not to be wondered at that students of children's dental conditions in the past have overlooked the fairly frequent occurrence in their mouths of the other types of periodontal disease.

Let us consider uncomplicated gingivitis first, however. It occurs with a wide variety of manifestations. The degree of redness varies or there may be no departure from the normal color of the gingival margin, the inflammation being indicated by thickening of the gum margin only. Hypertrophy as contrasted with thickening only is a fairly common finding. The variations mentioned do not, however, provide a suitable basis for a classification, in my observation, since there seem to be no characteristic etiologic or other factors associated with them. The simplest and most useful classification I have found is that of *generalized* and *localized gingivitis.*

In generalized gingivitis (Fig. 1), as the name indicates, the inflammation is generally distributed, or in other words, the gingivae throughout the mouth are inflamed. This is in contrast to localized gingivitis, but with other areas showing no involvement. The localized type may be difficult to differentiate from the generalized, if inflammatory areas are widespread, as often happens, but such distinction may usually be made if the mouth is examined with due care.

Generalized gingivitis, for our present purpose, may be stated to include not only chronic but also acute types. The latter may include Vincent's infections and also stomatitis, the latter an ulcerative condition having some points of resemblance to Vincent's gingivitis, but showing a streptococcus as the predominating organism, and being without the characteristic foul odor of the Vincent's case.

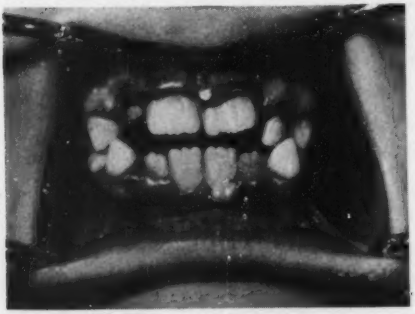


Fig. 1—Patient age 7. Generalized gingivitis. This type is usually associated with vitamin C deficiency.

As nearly as I can judge, generalized gingivitis almost invariably has a systemic background. This seems to be true even in the Vincent's cases. The systemic background is chiefly that of deficiency disease—a more or less severe deprivation of the vitamins, especially vitamin C, and, commonly, a mineral deficiency as well (Fig. 2). Since these deficiencies are usually associated with an acid-ash diet, there is a question as to whether this factor also plays a part. In all such cases addition of vitamin C plus minerals to the diet, or increase of amount, if already being given, will give gratifying results. May I suggest that if orange juice is selected as the source of vitamin C in a case of Vincent's infection or in stomatitis, it be taken through a straw and that the mouth be rinsed with an alkaline solution after. In these cases children may reject orange juice, unless this precaution is taken, as the gingivae are very sensitive to the fruit acid. Synthetic vitamin C will, however, give equally good results, and causes no local irritation. Be sure that enough vitamin C is taken.

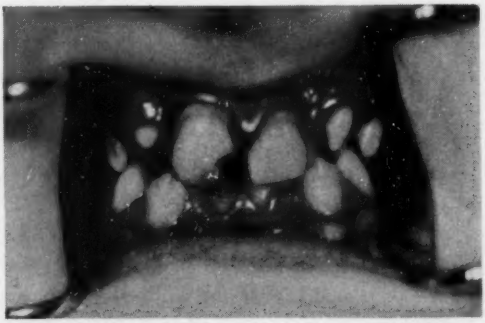


Fig. 2—Patient age 10. Generalized gingivitis. The condition of the gingivae suggests vitamin C deficiency. There is also the element of impaired function due to irregularity, with consequent lack of stimulation of the gingivae and periodontal structures. The tendency of the upper centrals to accumulate stain is due to a slight enamel hypoplasia; the accumulation of foreign material complicates the etiology of the gingivitis.

Since generalized gingivitis is nearly always associated with a state of health more or less perceptibly below par, the dentist will do well to consult with the family physician to at least apprise him of his findings and recommendations, at the same time suggesting an examination for chronic infection in the paranasal sinuses or tonsils, and a consideration of the adequacy of elimination.

Fig. 3—Patient age 10. Gingivitis of a somewhat generalized type with tendency to localization at areas of excessive stress. (The patient is not biting in centric occlusion, hence the stress on the upper left lateral is not indicated in the photograph.) Localized hypoplasia such as is shown here is usually due either to traumatic occlusion or to overlapping of teeth with consequent insufficient food scouring and the lack of gum stimulation associated with it.

Localized gingivitis is associated with some local factor causing direct irritation of the gingivae (Fig. 3). In this category we find the so-called "neglect" cases, those which are supposed to call especially for prophylactic treatment and improved home care. These cases show perceptible deposits of foreign matter, chiefly *materia alba.* And since the removal of this material and the maintenance of cleanliness will bring about a reduction of the inflammation, it is natural to stop at this point. What we should do, however, is to seek the reason for the initial accumulation of the accretion, for it has been my observation that the child's teeth do not get dirty entirely from lack of brushing. The two most common factors in the initiation of such deposits are sensitiveness, especially from caries, that inhibits mastication, and hypoplastic enamel.

Since caries itself may, if widespread, be looked upon as being due to a nutritional fault, there is some ground for discussion as to whether this type of gingivitis, so commonly associated with caries, is not also mainly systemic in its origin. I think there is much in that contention. Nevertheless it has been my experience that cases of what I call localized gingivitis respond readily to local treatment whereas the generalized cases do not, at least not so completely, thereby demonstrating the existence of the two types. The local treatment includes not merely prophylactic treatment, but the filling of all cavities, even the most minute pit cavities on occlusal surfaces, for all of these transmit sensation when food is masticated. Pain in chewing, even if but slight in degree, inhibits mastication and thus inhibits food scouring, the most effective tooth cleanser I know.

When caries occurs on approximal surfaces, opening up spaces between the teeth into which food can pack, we find a characteristic localized gingivitis at the point of impaction. The gingival papilla becomes swollen as well as red and is more painful than in other types of gingivitis.

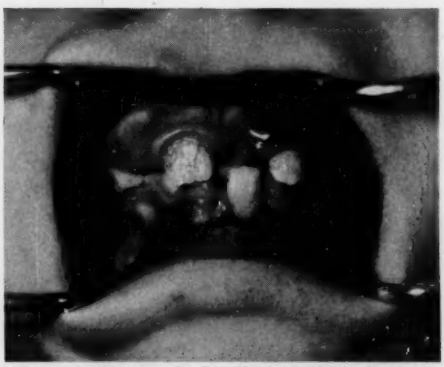


Fig. 4—Patient age 7. Localized gingivitis due partly to accretions on the tooth surface and partly to traumatic occlusion from the malposition. The foreign material on the surfaces of the upper incisors is retained by enamel slightly roughened because of hypoplasia. Note recession on the lower incisor.

Hypoplastic enamel in its gross form is familiar to all of you, but is, in that form, not common (Fig. 4). On the other hand, a mild degree of enamel hypoplasia, not so readily detected, is extremely common. This type of hypoplasia does not exhibit pitting or gross roughness; it is seen chiefly as a very slight roughness of the surface, presenting a very faint grittiness as the sharp pointed explorer is passed over it, or simply a dullness of the surface, a lack of gloss, seen when the dried surface is examined with a beam of light reflected on it. These conditions, to which attention has been called by Mrs. Mellanby,[4] are due to deficiencies in enamel formation. Enamel surfaces presenting such defects provide a splendid nidus for bacterial and mucinous accumulations. By the same token such surfaces are difficult to clean with the tooth brush, even in the most efficient hands. Hypoplastic enamel, then, provides the reason for the accumulation of foreign material found in many locations in the mouth and an explanation of the gingivitis found in such locations. Where such conditions are found, the operator should include in his prophylactic routine, procedures for the dressing down of the rough enamel and imparting to it a gloss that will facilitate home cleaning. D. D. Smith practiced and taught this more than thirty years ago.

Another type of localized gingivitis is that occurring around loose deciduous teeth. This is, of course, readily diagnosed. The gum becomes very red and seems to be on the verge of ulceration.

Somewhat different is the inflammation around slowly erupting teeth. In the anterior region the gum becomes thickened and is only slightly reddened, as a rule. In the molar region the tissue shows a more acute phase of inflammation, and ulceration may occur. Such inflammation, although localized around the erupting tooth, is nevertheless to be regarded as of systemic origin, since tooth eruption is a physiologic process and may and should take place without disturbance of gingival health. Vitamin C therapy will correct this type of inflammation while local treatment, although helpful, will fail to clear it up completely.

Periodontitis simplex is a disease cycle attacking the pericementum and alveolar crest along with the gingiva. In the adult it is either inflammatory, being the result of long undisturbed accumulations of calculus at the gingival line, or atrophic in character, the result of insufficient function together with possible systemic conditions of an as yet unidentified nature. In the child it is uncommon, since children seldom show much accumulation of salivary calculus. When such deposit is found, however, there is apt to be, with the usual gingivitis, a recession of the gingival border and some resorption of the alveolar crest. The atrophic type is also seen but is quite rare. The treatment of periodontitis simplex in the child consists chiefly of prophylactic treatment and instruction in home care of the mouth. Since formation of calculus in the child's mouth, at least before ten years of age, is unusual, its presence in any considerable quantity, with tendency to recurrence after cavities are filled and function restored, should lead one to suspect some systemic fault.

[4]Diet and the Teeth. Mrs. May Mellanby, Medical Research Council, London, 1930, 1934.

Periodontitis complex is often found in the child's mouth. It will be recalled that in periodontitis complex the dominant etiological factor is traumatic occlusion (Fig. 5). Too little attention has been paid to the presence of this source of irritation in the dentition of the young child. Also there has been virtually no attention given to disturbances other than gingival that are often found as a result of the abnormal stresses induced by this force.

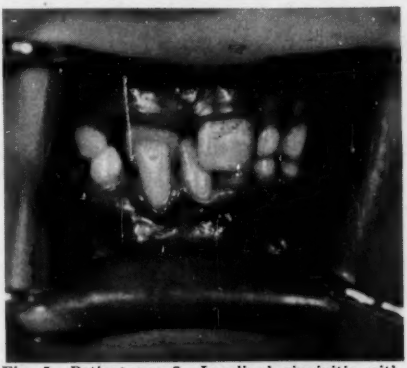


Fig. 5—Patient age 7. Localized gingivitis with recession on the lower incisors due to traumatic occlusion. Note the thickened gingival margin on each of the upper central incisors. The lower right central incisor was quite loose.

In considering this factor, we must constantly keep in mind the fact that traumatic occlusion affects first the pericementum. And since the pericementum extends from the gingiva to the tooth apex, the secondary effects of traumatic occlusion may be felt in either of those locations. Thus, in the child, we may and frequently do have pulp disturbance, often of severe degree, where stresses are in excess of normal. This will manifest itself in symptoms of pulpitis especially in the posterior teeth. These symptoms often develop some time after a filling has been placed, and the natural assumption is that the pulp was not adequately protected when the filling was inserted. This phenomenon occurs however even when all proper precautions have been taken. Examination of the occlusion in such cases will show excessive stress, sometimes due to a high spot on the filling, but quite as often a high spot on the tooth itself. In the latter case the explanation of the exacerbation after filling lies in the superimposition of the irritation caused by cavity preparation on a previously dormant pericemental irritation caused by traumatic occlusion.

Another disturbance due to traumatic occlusion, of which evidence can be found, yet which does not always involve the gingiva, is a slight alveolar resorption affecting the entire alveolar wall to a uniform extent. This can be detected in the X-ray as a thickening of the pericemental line. When it occurs it is to be found rather generally distributed throughout the mouth. It is most commonly seen in cases in which one or more first permanent molars have been lost and where the remaining teeth have been allowed to drift. Although the gingivae in these cases are often perfectly normal in color and form, the occurrence of the pericemental thickening leads inevitably to the thought that in these cases the foundation is already laid for a severe periodontoclasia later in life.

When the anterior teeth are involved by traumatic occlusion, there is apt to be a severe localized gingivitis, usually accompanied by alveolar crest resorption

and gingival recession. Mobility, sometimes of considerable degree, is usually present (Fig. 6). The only real difference between such cases and the cases of periodontitis complex in the adult, is that in the child pocket formation is extremely rare. The pericementum of the child, in other words, seems to have a very high resistance to destructive bacterial attack.

Fig. 6—Patient age 9. Localized gingivitis due to traumatic occlusion. The trauma is incident to the excessive overbite, this in turn being chiefly due to early loss of posterior deciduous teeth. The lower central incisors are slightly loose.

I have found little evidence so far to indicate any considerable or necessary connection between periodontitis complex and nutritional or other systemic shortcomings, except that, of course, there is always the possibility of a complication in any particular case. Treatment, therefore, is directed chiefly along local lines and includes improvement of occlusal relations by such means as may be needed or are feasible. Since the child does not have a Curve of Spee, occlusal disbalance due to excessive overbite of the anterior teeth cannot be relieved by shortening them, as is so often done with the adult. Distribution of stress among the teeth of this segment must suffice, the effort being made to insure that all the anterior teeth shall have equal stress in centric and protrusive positions (Figs. 7 and 8).

Before closing, I must say a few words about periodontoclasia in the adult. It has been a rather common assumption that this disease is characeristic of physical maturity and that we do not have to worry about it until the patient approaches middle age. I think we must now give thought however to the possibility that periodontoclasia, to a very real extent, has its origin in childhood.

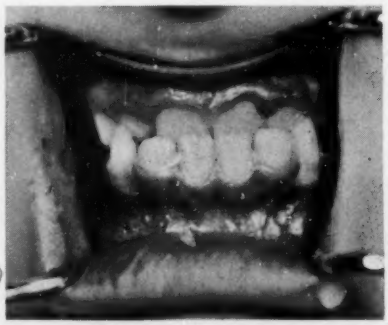


Fig. 7—Patient age 8. Localized gingivitis due to traumatic occlusion. The class III malocclusion shown in this case is an acquired condition resulting from the enforced habit of chewing food on the anterior teeth, the result of early extraction of deciduous molars. Note the thickening of the gum margin over the upper central incisors with recession over the left central. Note thickening of gingival margin on the lower incisors.

Much thought has been given to the origin of traumatic occlusion as found in the adult. It is quite generally agreed that malocclusion is apt to lead to traumatic occlusion and considerable attention has been given to the events that induce malocclusion. This has tended to center on events occurring subsequent to the sixth year, especially loss of the six-year molars. Without intending to draw your attention away from this very important cause of malocclusion,

I should like to call attention briefly to some events that may occur even earlier and that have quite as disruptive an effect. Enamel hypoplasia of the deciduous molars may be followed by disintegration of their occlusal surfaces and consequent closing of the bite. Premature loss of deciduous molars may result in drifting of permanent molars, loss of space in the premolar region with crowding of these teeth, and excessive overbite of the anterior teeth. Lack of jaw growth manifested before the fifth year and tracing either to diet deficiency or insufficient function may be an important and correctable cause of malocclusion and subsequent periodontal disease. It seems clear then that we should not only give more attention to periodontal disease in children's mouths, as part of the dental service due them, but should regard childhood and even the prenatal period, as the time for beginning the preventive periodontal program.

Fig. 8—Patient age 11. Localized gingivitis with recession, especially on the lower left central incisor. This condition is partly due to traumatic occlusion, but the extreme recession on the central points to an inferior alveolar structure which consequently rapidly wasted away under the influence of the trauma.

Infectious Mononucleosis*†

By JAMES E. COTTRELL, M. D.

Philadelphia, Pa.

THE disease which in this discussion is called "infectious mononucleosis" has been widely known by several other names. The commonest alternative designation, and the one which is usually used in Great Britain, is "glandular fever"; this name is derived from the term "Drüsenfieber," which Pfeiffer used in 1889 when he described the occurrence of the syndrome in children. Baader, in 1922, coined the name "Monozytenangina"; this, and its English equivalent "monocytic angina," are still sometimes found in the literture. Other designations are "infectious monocytosis," "acute lymphadenosis," and "acute benign lymphoblastosis." The writer prefers the name "infectious mononucleosis" for several reasons: first, it implies the infectious nature and the outstanding feature, the blood picture; second, the writer objects, even at the risk of seeming pedantic, to the name "glandular fever" because it refers to involvement of lymph-nodes, which are not glands at all, and no true glands are greatly affected; and third, because the name "infectious mononucleosis" has become well established in this country and is used in the Standard Classified Nomenclature of Disease.

*From the Medical Clinic of the Hospital of the University of Pennsylvania.

†Read before the American Academy of Periodontology, Atlantic City, N. J., July 8, 1937.

In this country, interest in infectious mononucleosis as a clinical entity dates chiefly from the reports of Sprunt and Evans (1920), Bloedorn and Houghton (1921), Longcope (1922), and Downey and McKinlay (1923). Since that time the literature on the subject, in American, British, and German journals, has grown too extensive to review here; two excellent recent reviews are those of Tidy (1934) in England, and of McKinlay (1935) in this country.

INCIDENCE: While not rare, infectious mononucleosis is by no means a common disease. This report is based upon 27 cases, admitted to the Medical Division of the Hospital of the University of Pennsylvania in the past 16 years; 12 of these cases have been reported previously by the present writer (1927). No intensive search was made for additional cases, and it is probable that others might be found in the records of the Student Health Service of the University, and of the nurses' infirmary of the Hospital. McKinlay, in 1935, had collected 50 cases over a period of 12 years from the Student Health Service of the University of Minnesota. Most of the reports are from institutions where considerable numbers of young persons are gathered under fairly close medical supervision, and describe the occurrence of the disease in students, pupil nurses, soldiers, and sailors. This may be partly an evidence of the epidemic tendency of the disease, and because of opportunities for the transmission of the infection in groups of young persons. It is more likely that infectious mononucleosis is observed so commonly in such groups because of closer observation and frequent routine examinations of the blood; and probably many cases run their course undiagnosed in the general population because of resemblance to common infections of the type of "grippe" and tonsillitis, and because the condition of the patient does not seem sufficiently serious to demand examination of the blood. The clinical features of the disease, however, are consistent enough that if the practitioner is acquainted with them and bears them in mind, he probably could usually make a tentative diagnosis of infectious mononucleosis, and then confirm or disprove it by hematologic and serologic tests. For reasons which will appear later, the correct diagnosis of this disease often enables the practitioner to contribute a great deal to the peace of mind of the patient and his family.

Infectious mononucleosis is essentially a disease of the first four decades of life, and especially of the second and third. It has been reported as early as the age of four months and as late as seventy years. It is probably rare in the first year, and is certainly rare after forty. The present series is from the adult medical service of the hospital only, and thus includes no cases in children. The recorded ages of our 27 patients are as follows: 16, 1 patient; 17, 1; 18, 2; 19, 6; 20, 1; 21, 3; 22, 4; 23, 3; 24, 2; 26, 3; and 35, 1. Males usually outnumber females in the reported series (our series, 23 males to 4 females; McKinlay, 1935, 38 males to 12 females; McAlpin, 1935, 16 males to 15 females; Nygard, 1932, 20 males to 13 females; Baldridge, Rohner, and Hansmann, 1926, 38 males to 12 females). This difference in sex-incidence is probably more apparent than real, as males usually predominate in groups from which the cases are reported.

There is no doubt that the disease is transmissible, but the contagiousness is slight. In any series of cases, a study of their time of occurrence shows evidence of an epidemic tendency; definite epidemics are reported by Tidy and Daniel (1923) and Nolan (1935); Tidy (1934) states that a wide-spread epidemic occurred in England in 1930. The occurrence of sporadic cases suggests the existence of a carrier state. The incidence is highest in the months when the incidence of respiratory infections in general is highest. Of our 27 cases, the onset of 18 was during the months January to May inclusive; only two cases began in the summer (June to August inclusive); the remaining cases were in the autumn and early winter. In McKinlay's (1935) 50 cases, the onset of only one was in the summer; but his patients were all university students, whom he could be unlikely to see at that time.

VARIANTS: There is a fair amount of variation in some of the clinical manifes-

tations of infectious mononucleosis. Tidy (1934) describes three distinct types: the "glandular," commonest in children, of moderate severity, with little inflammation of the throat, and with enlargement of the lymph-nodes as the outstanding feature; the "anginose," commonest in adolescents and young adults, with severe membranous inflammation of the throat; and the "febrile," commonest in adults, marked by a prolonged febrile course, persistent enlargement of the lymph-nodes, and relatively little inflammation of the throat. Our cases can scarcely be divided into clear-cut types, but there is a tendency for them to fall in two groups; (1) those of short duration, but fairly severe general and faucial manifestations, including cases with membranous angina; (2) the more prolonged, sub-acute cases lasting several weeks or a few months, with mild but persistent general symptoms, low-grade fever, and enlargement of the lymph-nodes, and with but slight inflammation of the throat. There is no clear difference in the blood picture in these two groups; the briefer, severer cases are more likely to show an increase in the total leukocyte count, and the more prolonged cases tend to show a normal or slightly diminished total leukocyte count, with an increased proportion of lymphocytes and monocytes and a decrease in both relative and absolute number of polymorphonuclear leukocytes.

ONSET: Because of the slight contagiousness of infectious mononucleosis and vagueness of a history of exposure in most cases, we are unable to deduce from our series the duration of the incubation period. It is variously estimated as from four or five days to three or four weeks. In the majority of instances the onset is fairly acute, and resembles that of grippe or mild influenza, with malaise, feverishness, headache, muscular aching, sore throat, and coryza. Cough is less frequent than coryza and sore throat. Photophobia, smarting of the eyes, and lacrimation occur fairly often. Six of our 27 patients had chills, and two had sweats without chills. In some cases enlargement of the cervical lymph-nodes is the first feature to attract the attention of the patient, preceding even malaise and sore throat by two or three days. Slight epistaxis occurs occasionally.

THE FAUCIAL, PHARYNGEAL, AND ORAL LESIONS: Subjective soreness of the throat is complained of by the majority of patients—21 of our 27. Most of those who do not experience soreness nevertheless show congestion of the throat. The objective changes may consist only of redness of the fauces, the tonsils if present, and the posterior pharyngeal wall. The pharyngeal lymphoid tissue is swollen, as well as the tonsils. In the severer cases, the uvula is often edematous. In twelve of our cases, exudate was present in the throat. This may vary from a few flecks on the surface of the tonsils to a membrane similar to that of diphtheria, covering the tonsils and extending onto the tonsillar pillars and the pharyngeal wall. The membrane is whitish or grayish, usually lighter in color than diphtheritic membrane and slightly less adherent; in some instances it is clinically indistinquishable from the latter. Ulceration is occasionally seen. Cultures from the throat lesions are, of course, negative for diphtheria, and show only the common mouth flora. Smears from exudative or ulcerative lesions are frequently positive for the spirilla and fusiform bacilli of Vincent (10 of our cases). In short, some degree of angina is an almost constant feature of the disease; this angina may range from slight redness of the throat, even without subjective soreness, to severe inflammation with edema, exudation, and ulceration.

The manifestations in the mouth proper are less striking. In one of our cases the buccal mucosa was diffusely reddened; in two there were one or two small aphthous ulcers on the buccal mucosa; in three there were a few petechiae on the roof of the mouth or the soft palate; in one there was, at the onset of the infectious mononucleosis, inflammation of a partially impacted third molar, probably a mere coincidence. In the series of Baldridge, Rohner, and Hansmann (1926), 26% of the patients complained of sore and bleeding gums, and smears from the gums occasionally show Vincent's organisms. One never sees, however, the severe gingivitis and stomatitis with bleeding and necrosis

that occur in acute leukemia and in so-called agranulocytic angina.

THE LYMPH-NODES: Enlargement of lymph-nodes is a constant feature of the disease, and is usually seen first and in most marked degree in the cervical region. The posterior cervical chain is usually most involved; the "peri-tonsillar" nodes, under the angles of the mandible, are usually affected; the anterior cervical chain and the supraclavicular nodes enlarge in many instances. Sometimes the pre-auricular and post-auricular nodes are felt, but not the occipital group. The nodes vary in size from those barely palpable to those comparable in size to a walnut. They are discrete, and firm in consistency or only slightly softened. They are frequently slightly tender, but never exquisitely so. In the acute cases with severe exudative angina, the surrounding tissues are often edematous and the over-lying skin slightly reddened; in these cases the marked enlargement of the nodes together with the edema sometimes gives the neck a pyramidal contour. The nodes do not break down and suppurate. Frequently the involvement of cervical nodes is predominantly unilateral, with only a few small ones palpable on the opposite side. There is a considerable variation in the time of enlargement of the lymph-nodes; in a few cases it is the first feature detected, in most it appears in from one to three days after the onset of general symptoms, and in the sub-acute cases it may not appear until the second or third week of the disease and is accompanied by a secondary rise in temperature. In the cases with little inflammation in the throat, the degree of enlargement of nodes is strikingly disproportionate to the angina.

Involvement of other lymph-nodes usually follows a short time after enlargement in the cervical region but is less striking. In our series enlargement of axillary nodes was noted 17 times, of inguinal and femoral nodes 15 times, of epitrochlear nodes 11 times. In any of these regions the enlargement may be unilateral; or a single node may be palpable in each axilla. It is probable that mediastinal, mesenteric, and other internal lymph-nodes are not infrequently involved. We have not seen abdominal symptoms of any marked degree, but their occurrence in some reported series suggests that other lymphoid structures, such as Peyer's patches and the lymphoid tissue of the appendix are also swollen. In an occasional case which is otherwise typical, involvement of lymph-nodes may be very slight; one of our patients showed only a very few small cervical nodes during the entire course, and did not have a palpable spleen.

In the brief, acute cases, the nodes diminish rapidly in size with subsidence of throat inflammation, fever, and general symptoms, but slight enlargement usually persists for several weeks after the patient is subjectively well. In the sub-acute cases, the enlargement may persist for many months.

The histology of the lymph-nodes has been reported many times (Fox 1927, Downey and Stasney, 1935). The changes consist principally of diffuse hyperplasia of lymphoid and reticulum cells, with disarrangement of the architecture of the node. The histologic picture is said to be readily distinguishable from those of leukemia, sarcoma, Hodgkin's disease, and so forth.

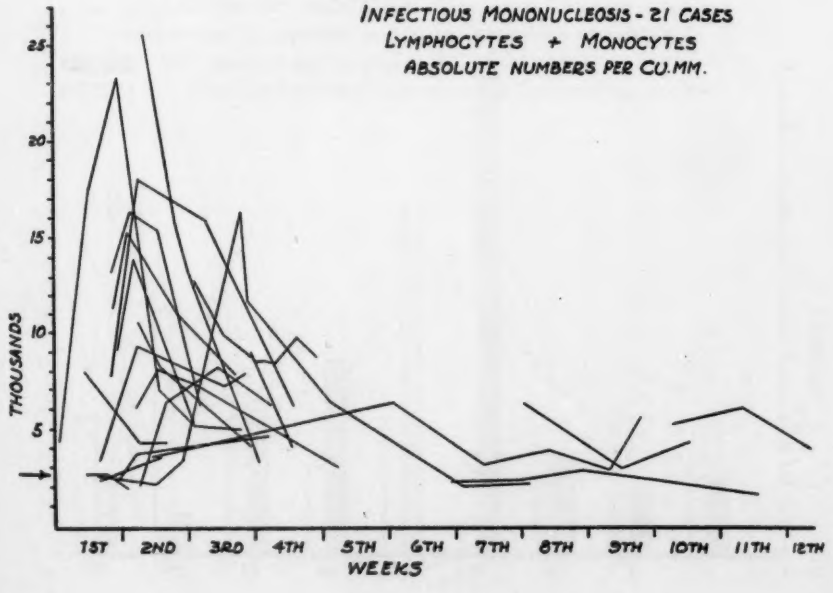
THE SPLEEN was palpably enlarged at the time of admission, or of first examination, in 13 of our cases. In one the spleen was first felt about the twentieth day of the disease. In some, enlargement of the spleen was not detected at any time, in spite of repeated and careful search for it. It is possible that in some cases splenic enlargement is transient, and escapes attention. It is safe to say that splenic enlargement may be expected at some stage of the disease in well over half of the cases. The enlargement is moderate, the size never approaching that found in leukemia; the spleen is firm but not excessively hard, maintains its shape, and is sometimes slightly tender. The enlargement of the spleen is of shorter duration than the enlargement of lymph-nodes, and frequently the spleen has receded to normal size before the subjective symptoms have disappeared. Gordon (1935) mentions one case in which enlargement of the spleen was present, with almost no enlargement of lymph-nodes.

THE RASH: Two of our patients exhibited a diffuse morbilliform rash on the trunk and extremities. Three had a sparse rash of pinkish macules, resembling the rose spots of typhoid fever. One had a transient scarlatiniform blush over the thorax. These first two varieties represent the types of eruption to be expected in a small proportion of cases, appearing in our cases between the fifth and tenth days, and lasting only a few days. Eruptions are not a prominent feature in series reported in this country; but Tidy (1934) states that he observed a rash in nearly every adult case of his "febrile" type of the disease. Purpuric eruptions appear to be very uncommon. One of our patients had numerous petechiae on one arm; Williams (1931) reports his own case as having been marked by many purpuric spots, especially over the legs.

THE BLOOD-PICTURE: Here is seen the characteristic feature of the disease—that is, the striking increase in the number, both relative and absolute, of the mononuclear leukocytes. There has been much debate among hematologists as to the morphology of the principal cells, and even as to which group of leukocytes they belong to. The consensus, at present, appears to be that the predominant cells may consist of any variety of lymphocytes of any age except the youngest lymphoblast forms, and of monocytes (the endothelial leukocytes) as well. The proportion of these types of cells varies in different cases; occasionally other forms, such as histiocytes and Turck irritation cells, may appear. Downey, whose article of 1923 is the classic study of the hematology of this disease, believes that the blood-picture can be distinguished from that of leukemia by the cell-morphology alone; other hematologists are not in complete agreement with this opinion. Good recent reviews of the technical hematologic aspects are those of Nygard (1932) and Downey and Stasney (1935).

The total leukocyte count is increased at some stage in the disease in the majority of cases. There appear to be a few cases in which the total count does not rise appreciably, the change in the differential count being due to suppression of the polymorphonuclear leukocytes with relative, or only slight absolute, increase in

FIG. 1



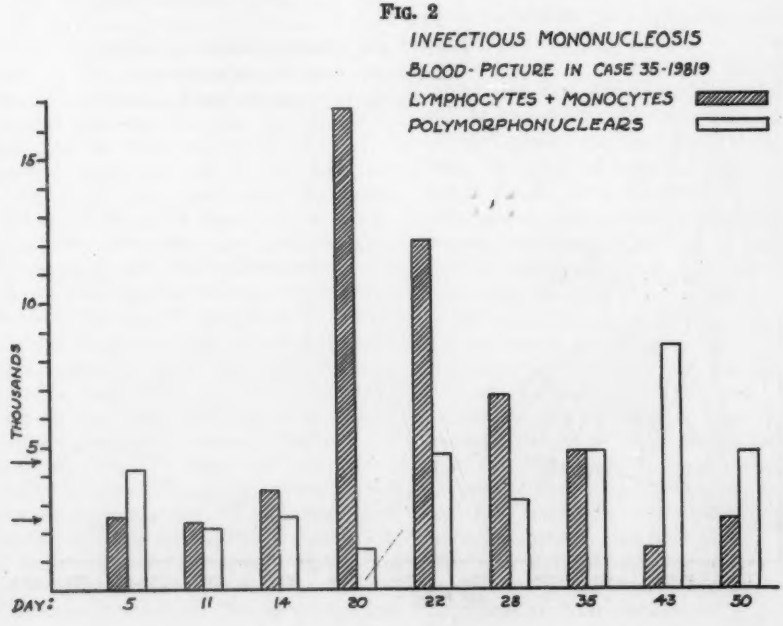
the mononuclear cells. The leukocyte picture changes rapidly from day to day, and the peak of the total count might readily be missed unless frequent examinations are made.

The increase in the total leukocyte count is usually moderate; in 17 of our cases the highest figure was between 10,000 and 18,000; there were two counts of about 22,000, and two of 31,000, which is the highest figure in our series. In general, the top level for the total leukocytes in infectious mononucleosis may be considered to be about 30,000 per cu. mm. Occasional cases with slightly higher counts are reported, for instance, McAlpin's (1936) case with 42,000. On the other hand, in six of our cases the highest counts were between 5,000 and 10,000; higher counts may, of course, have been missed because the patients did not come under observation at the proper stage of the disease. The lowest total count we have recorded is 4,600, and the greatest drop was from 31,000 to 5,200. Rosenthal (1932) has recorded a number of cases in which the total count was distinctly subnormal, ranging from 900 to 4,200, but with a high proportion of mononuclear cells; he calls this syndrome "leukopenic infectious monocytosis".

There is adequate evidence that the mononucleosis, with little or no polymorphonuclear reaction, is a specific effect of the infecting agent, not an individual peculiarity of the patients in responding thus to an infection. There are many reported instances in which polymorphonuclear leukocytosis has occurred, in response to some of the usual stimuli, in patients who had previously had infectious mononucleosis.

The writer feels that the mononuclear cells (lymphocytes plus monocytes) must reach a percentage of at least 40% at some stage of the disease to justify a diagnosis of infectious mononucleosis; they usually attain a much higher level than that—in most of our cases, 60% to 85%. Not infrequently the percentage of mononuclear cells may reach 90%, as it did in three of our cases. Usually, but not always, this change in the differential count is detected in the first examination.

FIG. 2



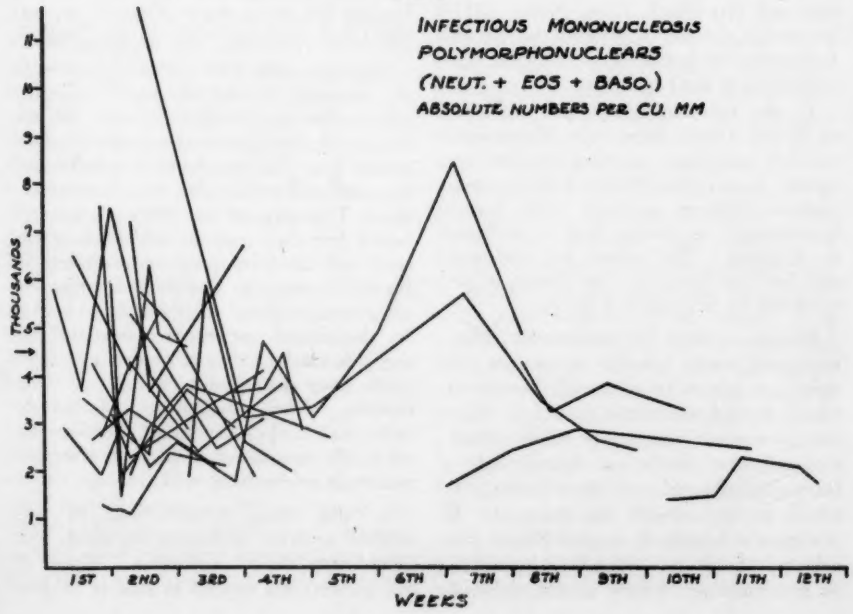
The total count and the mononuclear percentage are likely to be highest in cases with marked angina, to reach their peak when the angina is at its height, and for a time to decline with the fever and general symptoms. The blood count does not return to normal with symptomatic recovery, however; the changes persist for weeks or a few months, but eventually disappear.

Changes in the polymorphonuclear leukocytes are less striking, but are noteworthy. A consideration of the absolute numbers shows that there is often a slight increase during the course. In many cases, however, there is a distinct decrease as the mononuclears rise; in a few of our cases the number of polymorphonuclears fell below 2,000 per cu. mm. This circumstance has been commented upon previously (Cottrell, 1927) as suggesting a relation between infectious mononucleosis and the syndrome of agranulocytic angina. It is so striking in Rosenthal's (1932) leukopenic cases that he refers to that syndrome as "a benign form of agranulocytosis".

Anemia is not a part of the picture of infectious mononucleosis. In the prolonged sub-acute cases a very slight secondary anemia may develop, but it is never severe. It is noteworthy also that the blood-platelets are not diminished, and that (as stated above) purpuric manifestations are almost negligible. This may be a point of importance in differentiating certain cases of infectious mononucleosis from acute leukemia (Pepper and Farley 1933).

THE SEROLOGIC REACTIONS: In mammalian blood, including human, there are natural agglutinins and lysins for the erythrocytes of heterologous species although the animal has never been immunized to the erythrocytes in question. Because these substances are not produced in response to a specific antigen, they are called "heterophile" antibodies. It has long been known that the injection of horse serum produces in the human heterophile agglutinins and lysins for sheep erythrocytes, in addition to those which occur naturally. In the course of some other work Paul and Bunnell (1932) found

FIG. 8



that the blood-serum of patients with infectious mononucleosis agglutinated sheep-cells in high dilutions, and suggested that this phenomenon might prove to be of service as a specific diagnostic test. Further observations by many workers have demonstrated that this "heterophile antibody test" or "Paul-Bunnell test" is positive in high dilutions in a considerable proportion of cases, and that agglutination occurs only in low dilutions in the great majority of normal cases and in other diseases. It is usually possible to recognize, by the history, the high titre produced by the recent injection of horse serum. Stuart, Welch, Cunningham, and Burgess (1936) state that it is possible, by methods based on selective absorption of the agglutinins, to distinguish between the normal heterophile antibodies, those produced by injection of horse-serum, and those occurring in infectious mononucleosis.

This test has been made in only three of our cases, and was not especially helpful in those, as it was positive only in the lower dilutions (1 to 32 and 1 to 64). McKinlay (1935) found the test positive in 5 of 10 cases in which it was done. Most reports (Stuart et al. 1936; Weintein and Fitz-Hugh 1935; Nolan 1935) imply that the test is constant and specific. Evidently further experience with it is needed for a final evaluation.

In the sub-acute cases (corresponding to Tidy's febrile type) the Wassermann reaction sometimes becomes positive, but reverts to negative by the time the temperature becomes normal. This feature is important, as it may lead to confusion in diagnosis. The writer has not seen, and has not found in the literature any reference to, a positive Kahn test.

MISCELLANEOUS EXAMINATIONS: Albumin and casts usually appear in the urine, as might be expected; hematuria, which is said sometimes to occur (Tidy 1934), was not seen in our cases. Blood-cultures were sterile, or showed only a few staphylococci or diphtheroids, to which no significance was attached. In one case, a hemolytic staphylococcus was cultured from an excised lymph-node. As in almost any febrile disease, sedimentation of erythrocytes is accelerated. One of our patients had had a mild attack of malaria shortly before the onset of infectious mononucleosis, and plasmodia had been found in his blood at that time; the blood was searched for malaria during the course of infectious mononucleosis in a number of our cases, always with negative results. Other laboratory examinations made without significant results include: reticulocyte percentage; fragility of erythrocytes; van den Bergh reaction; Felix-Weil reaction; agglutinations for typhoid, para-typhoid, and brucella abortus; blood urea and blood sugar; pressure, cell count, sugar, and globulin of the cerebrospinal fluid.

TEMPERATURE: The temperature is irregular, but not of the septic type. During the height of the disease, it usually ranges between 100° and 101° F., but is likely to be higher in the cases with marked angina and leukocytosis, reaching 102° or 103°. We have only once seen the temperature as high as 104°. The period of high temperature is usually brief, and is followed by a longer period of low-grade fever. In the sub-acute cases, irregular temperature, ranging from sub-normal to between 99° an d100°, may be seen for many weeks. One of our patients had no fever while in the hospital.

COURSE AND PROGNOSIS: In general, the severity of the disease is comparable to that of a mild influenza. At the height of the disease the patients often appear very ill; the throat is painful and the swollen lymph-nodes very uncomfortable. This stage of maximum discomfort lasts a few days, and the subjective symptoms and the fever gradually subside. In the brief cases, the duration of subjective symptoms is about two or three weeks; in the longer sub-acute cases malaise, slight headache and sore throat, and low-grade fever may persist for two or three months. The enlargement of lymph-nodes, and the abnormal blood-count, ordinarily remain for some time after the patient is subjectively well.

In this series complications of any marked severity have not occurred, and indeed are almost unknown. None of our patients has seemed so sick as to give

rise to fear of a fatal termination, and all have recovered completely. There are reports of a few deaths from bronchopneumonia, secondary streptococcic infection with sepsis, or a pre-existent disease such as chronic nephritis. All writers agree that so far as the disease itself is concerned, the prognosis is invariably favorable. That infectious mononucleosis does not imply impairment of an individual's subsequent health is indicated by Hall (1934) who stated that his original patient had not had a day's illness since the attack nineteen years previously. The writer is still acquainted with several of our earlier patients, who have been well and working hard for eight to thirteen years since the disease.

DIAGNOSIS: In view of the benign nature of infectious mononucleosis, it might almost be said that the principal importance of making the diagnosis is to enable one to reassure the patient and his family, and to set one's own mind at ease in regard to the management of the case. The disease most likely to give rise to confusion is acute lymphocytic leukemia, and in view of the malignancy of this disease the importance of differentiation is both great and obvious. In leukemia the total leukocyte count is usually far in excess of the figure of 30,000 to 40,000 which is rarely if ever exceeded in infectious mononucleosis; leukemia is marked by anemia, thrombocytopenia, purpura, and progressive deterioration of the patient; and Downey (1923 and 1935) feels sure that a differential diagnosis can be made by the cell morphology alone. In a doubtful case, a normal platelet count is the strongest argument, from the hematologic aspect, against a diagnosis of leukemia (Pepper 1937). Some cases with low leukocyte counts may be difficult to distinguish from agranulocytic angina, but in the latter condition the leukopenia is extreme, the leukocytes of the granular series almost entirely disappear from the blood-stream, the inflammation of the mouth and throat is more severe, the toxemia is profound, and the gravity of the illness obvious. Downey and Stasney (1935) state that they have seen one case in which a blood-picture typical of infectious mononucleosis later changed to agranulocytosis with 800 leukocytes and with fatal termination. Acute follicular tonsillitis, simple or tuberculous inflammation of cervical lymph-nodes, and other conditions with local manifestations suggesting those of infectious mononucleosis, are readily differentiated by the blood-picture. Infectious diseases accompanied by lymphocytosis—for instance, pertussis—are distinguishable by their characteristic clinical features. The heterophile antibody test may prove to be a valuable aid in confirmation of the diagnosis. However, it is usually possible to make the diagnosis easily and with certainty by careful consideration of the symptoms, physical signs, and blood count. It can be said that none of our cases has offered serious difficulty in diagnosis, when once the staff of the medical division of the hospital had become familiar with the syndrome from having observed a few cases.

ETIOLOGY: While it is obvious that this syndrome is of infectious origin, the causative organism has not been surely identified. The frequent association of the spirilla and fusiform bacilli of Vincent with the ulcerative and exudative lesions in the throat gave rise to the suspicion that these organisms were the cause. However, so many cases, typical in course and blood-picture, occur without the presence of Vincent's organisms that this argument fails; and one not infrequently sees Vincent's infection of the tonsils with polymorphonuclear leukocytosis instead of mononucleosis. Gorham, Smith, and Hunt (1929) reported that they produced the blood-picture of infectious mononucleosis in guinea-pigs by inoculating the animals with membrane from the throat of a patient with the disease, and also with two strains of spirilla isolated from the throats of patients. Cultures of the fusiform bacilli, diphtheroids, and several other organisms failed to produce similar results. In the experiments with pharyngeal membrane other materials than Vincent's spirilla were obviously injected into the animals; in the case of the artificially grown spirilla, one may wonder if some other infective agent was transmitted along

with the spirilla from culture to subculture to animal.

The literature contains several other tentative identifications, made on the basis of animal experiments, of a causative organism. Coon and Thewlis (1923) isolated a diphtheroid from an excised lymph-node, and state that this organism produced the disease in guinea-pigs. The culture of diphtheroids from the throat, the blood, and the lymph-nodes has repeatedly been reported; these ubiquitous organisms are highly variable in their characteristics, and usually of low pathogenicity. Nygard (1932) incriminates the "bacterium monocytogenes hominis", which he cultured from patients' throats. Bland (1931) suggests that a protozoan organism of the genus Toxoplasma may be the cause; his work was based on the inoculation of rabbits and monkeys with patients' blood.

None of these observations has been adequately confirmed. The diversity and the somewhat unconvincing nature of the bacteriologic evidence naturally suggests to the clinician that the causative agent may be a filtrable virus. Acute lymphocytic leukemia presents a number of features which arouse the suspicion that it may be a virus disease; it has previously been suggested (Cottrell 1927) that the virus responsible for infectious mononucleosis may prove to be related to the hypothetical one causing acute leukemia. It is also possible that under varying circumstances, more than one virus or microorganism may be capable of producing the picture of infectious mononucleosis.

TREATMENT: This, fortunately, is the simplest aspect of the subject. For the most part it is symptomatic, such as would be indicated in any mild infection; nothing is known which is able to shorten the course of the disease. The severe throat lesions, particularly when smears are positive for Vincent's organisms, deserve special attention. In those cases the most effective gargles are the oxidizing ones, such as hydrogen peroxide and sodium perborate; we have also used local applications of neo-arsphenamine, 5% solution in glycerin. In some stubborn cases, neoarsphenamine intravenously seems to hasten healing. In the cases without membrane in the throat and without Vincent's organisms, mild antiseptic gargles such as hot metaphen solution, 1-8000 to 1-5000, may lessen the angina.

SUMMARY

Infectious mononucleosis presents a clinical and hematologic picture sufficiently definite to justify considering it an entity.

While the disease is obviously infectious, the causative organism has not been identified with certainty; it may be a filtrable virus, or more than one micro-organism may be capable of producing the disease.

The salient features of the disease include:

1. General symptoms of greater or less severity, as in any infectious disease.

2. Inflammation of the throat, ranging from mild congestion to severe exudative and ulcerative processes.

3. Increase in the mononuclear leukocytes (lymphocytes and monocytes) to 40% or more of the total leukocytes; this feature is essential for a diagnosis. Usually, but not always, the total number of leukocytes is increased; there may be a temporary diminution in the absolute number of polymorphonuclear leukocytes.

4. Enlargement of lymph-nodes.

5. Enlargement of the spleen in over half of the cases.

The course may be acute and brief (two or three weeks) or sub-acute and prolonged (several months).

The prognosis is invariably good, complications almost unknown, and management simple.

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Report of a Study on the Etiology of Bleeding Gums*

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WITHOUT question, bleeding from the gingival tissues is a common finding having a high incidence among patients. The causes are numerous and may indicate, as a symptom, a mild dysfunction easily cleared up, or a serious condition necessitating vigorous treatment. This study concerned itself with the etiology of the bleeding as a whole (Figures 1, 2), with primary emphasis placed on the effect of latent or chronic deficiency in vitamin C as a factor[1] [2] and the various changes in the gingival tissues that occur with a controlled type of treatment.

While local effects of diet on the teeth and supporting structures have long been accepted as valid, considering such influences as hard and soft foods, detergent and non-detergent diets, decalcification effect of sweets[3] etc., only the great increase in the modern knowledge of cevitamic acid, its properties and influences,[4] and the effects of its demonstrable absence or suboptimal quantity in the diet (or difficulties in absorption), bring to light the influence it has orally and in relation to the general health of the patient.[5]

* Read before the American Academy of Periodontology, July 9, 1937, at Atlantic City, N. J.

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[2] This investigation was made possible through the establishment of a research fellowship by the Bristol-Myers Co.

CLASSIFICATION OF ETIOLOGY OF BLEEDING GUMS

FIG. 1

PRIMARY EXTRINSIC FACTORS (External Influences)

1. Injury
   - A. Acute
     1. Severe cut or burn
     2. Toothbrush trauma
     3. Vincent's infection
     4. Idiosyncrasy to food or drug
   - B. Chronic
     1. Calculus
        - a. Salivary
        - b. Serumnal
     2. Food impaction
     3. Toothbrush trauma
     4. Gingival irritation by dental restoration
     5. Abnormal occlusal habits
     6. Traumatic occlusion
     7. Non-occlusion
     8. Cervical dental caries
     9. Mouth breathing
     10. Oral habits
     11. Non-detergent diet
     12. Occupational hazard

FIG. 2

PRIMARY INTRINSIC FACTORS (Internal Influences)

1. Blood dyscrasias
   - A. Affecting the blood cellular elements
     1. The leukemias
        - a. Chronic myeloid (or myelocytic)
        - b. Chronic lymphatic (lymphocytic)
        - c. Acute myeloblastic
        - d. Acute lymphatic
        - e. Unusual forms as aleukemic leukemia, chloroma, etc.
     2. The anemias
        - a. Pernicious (Addison's)
        - b. Secondary anemia
        - c. Acute aplastic anemia
   - B. Affecting the blood clotting elements
     1. The purpuras
        - a. Purpura hemorrhagica
     2. Hemophilia
2. Changes in the blood vessels
   - A. Scorbutic conditions
     1. Acute
     2. Latent, or chronic scurvy
   - B. Arteriosclerosis
   - C. Protein deficiency
   - D. Allergy
     1. Focal infection
     2. Schonlein—Henoch's purpura

SECONDARY INTRINSIC FACTORS

(Internal Predisposing)

1. Faulty histological structure of the gingival tissue
2. Diabetes mellitus
3. Endocrine disturbances
   - a. Pregnancy
   - b. Vicarious menstruation
   - c. Endocrine changes in adolescence
   - d. Ovarian disturbances
4. Febrile states
5. Toxic states
6. Gastro-intestinal disturbances

SCURVY AND ITS HISTORICAL BACKGROUND

The clinical disease known as scurvy presents three outstanding symptoms:[6]

1. The gums become spongy, swollen and bleed easily.
2. Petechial hemorrhages are found especially in the hair follicles of the lower extremities, and bleeding occurs from the stomach, intestines, kidneys, nose, etc.
3. Subperiosteal hemorrhage and hemorrhage into the muscles.

Observation on individuals, corroborated by animal experimentation, have definitely determined this disease to be due to a deficiency of vitamin C, and its manifestations can, in most cases, be cleared up by its administration in the diet.[7] This condition frequently found fatal termination until almost modern times, causing terrible suffering, especially among sailors on long sea voyages, who lived on restricted diets in which this vitamin was absent. A method of trial and error was resorted to in an endeavor to find a source which would be efficacious. The literature is replete with examples where such additions i. e. orange juice, limes, fresh vegetables, concentrates, etc., were either partially or entirely satisfactory.

The establishment of the biological test for vitamin C gave real impetus to the study. Doctor Szent-Gyorgyi[8] in 1928 found by means of this test that the adrenal glands of oxen were rich in this substance, and by fractionation could yield a chemical called hexuronic acid, with the formula $C_6H_8O_6$, which he found would protect experimental animals against this disease. Doctor Tillmans[9], City Analyst of Frankfort, Germany, found an indigo blue coloring matter which was decolorized by fresh lemon juice but was not affected by imitations of stale juice, that is, it had reducing properties. In 1932, Waugh and King, chemists at the University of Pittsburgh, finally succeeded in

isolating vitamin C from lemons, found it to be a reducing substance and identical with Doctor Szent-Gyorgyi's hexuronic acid. The same year saw the chemical synthesis of this substance carried out by Reichstein and Grussner[10], chemists of Zurich, Switzerland, with the formula established as the lactone of three-3-keto hexonic acid.

PHYSIOLOGICAL PROPERTIES OF VITAMIN C

Cevitamic acid is absorbed into the blood stream from the stomach and intestines and so carried to the various cells of the body, acting there as an oxidizing-reducing substance, able, because of its chemical structure, to yield oxygen to an environment that needs it, and take up other elements in exchange. Thus, if oxygen is needed by the cell protoplasm, vitamin C gives up some of its own oxygen, but the process is also reversible, for when there is too much oxygen in any environment, the excess can be taken up and incorporated in its own chemical structure. It is in such consideration, as an oxidizing-reducing substance, that this vitamin assumes vital importance in the metabolic processes of every cell, its health and proper functioning.

Cevitamic acid favors the formation of a cementing substance which adds consistency to the blood vessel wall[11], and acts on the capillaries to prevent blood from flowing into the surrounding tissue. Lack in the supply of the vitamin leads to a corresponding increase in the permeability of the capillary walls, the fundamental fact upon which one type of test for deficiency, called the capillary resistance test, is based. The property to restore the integrity of the cellular elements of the wall by restoring and maintaining the intracellular cementum[12], and promoting normal viability of the individual cell has led to its use in treating various dyscrasias.

PRESCORBUTIC CONDITIONS

As modern interest in scorbutic manifestations developed, various evidences of impaired health were traced to suboptimal feeding or faulty absorption of vitamin C leading to a condition known as latent or subacute scurvy. In contradistinction to the clinical entity of scurvy proper, a prescorbutic condition may be evidenced subjectively solely by a vague lack of physical well-being, while lack of normal growth, anemia, tendency to bleeding from the gums, and general mild hemorrhagic tendencies are more specific symptoms[13].

Sufficient amounts of vitamin C may be taken in the diet to prevent the typical clinical picture of the deficiency disease from developing, but the full needs of the body for the vitamin may not be supplied. Gastrointestinal upsets of various kinds may act to inhibit the absorption of vitamin C in those cases where seemingly sufficient amounts are supplied in the diet but deficiency still exists. Another phase of this same general consideration should include those persons whose diet contains no foods which are commonly accepted to be excellent or even good sources of the vitamin but because of the larger amounts of poorer sources included in the diet, no deficiency will become evident: the need of the individual, and total amount, taken from whatever source, must both be considered.

Distribution of vitamin C in man is strikingly similar to the distribution in experimental animals. Those animals which require a direct dietary source of the vitamin (mice, fowl, etc., seemingly are able to synthesize it) the time required for marked general tissue depletion is apparently much shorter than the time required for symptoms of scurvy to appear. It is recognized that there may be some margin between a "state of unsaturation" as indicated by a failure to excrete vitamin C when a test dose is fed, and that fuller degree of deprivation which is needed to bring on the definite harmful effects of the vitamin deficiency.

METHODS USED IN THE INVESTIGATION

The various methods described in the literature for the determination of vitamin C deficiency[14 15 16 17] were investigated and those found most satisfactory were employed. These included the use of both a fixed positive (Gothlin method)[18] and a fixed negative pressure on the arm surface (Resistometer)[19] to cause capillary rupture and petechial formation, and the

determination of the excretion rate by titration with 2:6 dichlorphenolindophenol[20]. Methods of gingival biopsy, observations on the gingival tissues with capillary microscopy[21] and the use of the Resistometer to cause capillary hemorrhage on the gingiva and the inner surface of the lower lip were also applied.

1. *The Gothlin Test*[22]. The procedure includes the application of a sphygmomanometer to the upper arm at a constant pressure of 50 mm. for a period of fifteen minutes, the arm being held quietly at approximately the level of the heart (Fig. 3). Macroscopic petechiae appearing in an area of 60 mm. diameter at the center of the bend of the elbow are then counted, being made more evident by compression under glass. The normal limits have been determined and reported in the original paper[22] as being under 5 at 50 mm. pressure. Determinations of over 8 petechiae indicate deficiency.

2. *Resistometer on the arm.* This procedure includes the application of a negative pressure in a 1 cm. area by means of a simple apparatus[24] to the forearm below the bend of the elbow (Fig. 4). The recommendation is made[25] that the negative pressure be applied to the point where macroscopic petechiae first appear. In applying the test, however, experimentation showed it to be difficult to follow and a new set of standards were devised. Negative pressure is applied 3 cm. below the bend of the elbow, rapidly increased to a reading of 45 cm. as determined by the manometer on the apparatus and allowed to remain constant for a period of 10 seconds. The 1 cm. glass cup is then removed and the number of macroscopic petechiae counted under compression with glass. This standard test was used in the investigation.

3. *Resistometer on the gingiva.* Application of the Da Silva-Mello apparatus or Resistometer is attempted for direct readings of the capillary resistance of the gingival tissue. Glass applicators of various sizes and forms were tested and one made with an orifice of 1 mm. diameter, surrounded by an inverted lip of 1 mm. was found to be best suited. The glass applicator is placed in position on the gingival surface at the interproximal papilla, a negative pressure of 45 cm. exerted, and held in position for a period of 10 seconds (Fig. 5). The area is then compressed with glass, the tissue blanched, and examined for petechiae.

4. *Resistometer on the inner surface of the lower lip.* The mucous membranes of the mouth offer an interesting field for capillary resistance study. A glass applicator was devised having an orifice of 2 mm. diameter and an inverted lip of 1 mm.

FIG. 3

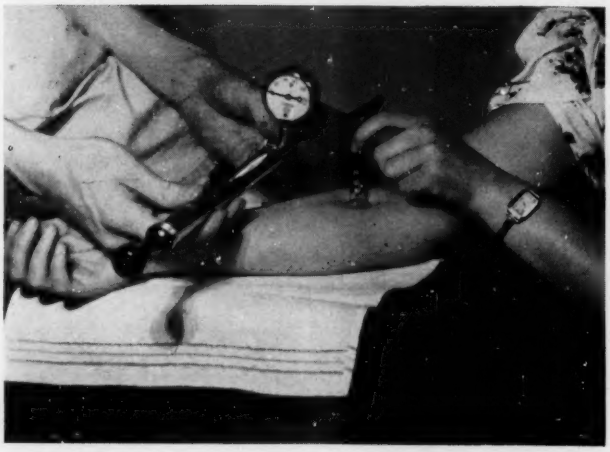

FIG. 4

width, for use with the Da Silva-Mello apparatus (Fig. 6). The tongue side of the inner surface of the lower lip was the site tested.

CHOICE OF PATIENTS

The patients were selected from those presenting for treatment at the Periodontia Clinic of the New York University College of Dentistry. Where bleeding was a symptom, efforts were made to arrive at its etiology, and the possibilities of a dietary deficiency in vitamin C were considered. Other phases of the clinical syndrome of latent deficiency were also entered into, such as the tendency to "black and blue" spots on relatively slight trauma, tendency to tire easily, nature and character of bleeding in the mouth and elsewhere in the body[26], anorexia, lack of resistance to infections, etc.

While vitamin C is the only known dietary factor that can influence the intrinsic power of the capillary to resist hemorrhage, effects of particular proteins are being investigated[27]. Complicating conditions that must be considered include various dysfunctions:

Factors Influencing Capillary Resistance (After Wiemer[28]):

1. Factors directly damaging the endothelium.

   a. Poisons, opium, neoarsphenamine, carbon monoxide, etc.

   b. Toxins such as scarlet fever, diphtheria, polyarthritis, etc.

   c. Metabolic products as in uremia and acetonemia.

   d. Scurvy.

2. Factors indirectly affecting the endothelium or capillary tonus.

   a. Physiologic variations as in menstruation.

   b. Endocrine diseases as exophthalmic goiter.

   c. Diseases of the spleen and reticuloendothelium system.

FIG. 5

The various factors besides the specific vitamin deficiency which lead to increased capillary permeability were ruled out by a blood examination including red cell count, white cell count, hemoglobin determination, differential white cell count, bleeding time and clotting time, a urinalysis, and, if necessary a complete physical examination by the patient's physician. Difficulties with absorption of cevitamic acid such as occur particularly in diabetes, gastric, and peptic ulcers, and other gastrointestinal upsets were contraindications in the choice of the patient. Frank or manifest scurvy is today not commonly encountered, but the incidence of oral gingival manifestations of latent scorbutic conditions has not been satisfactorily explained.

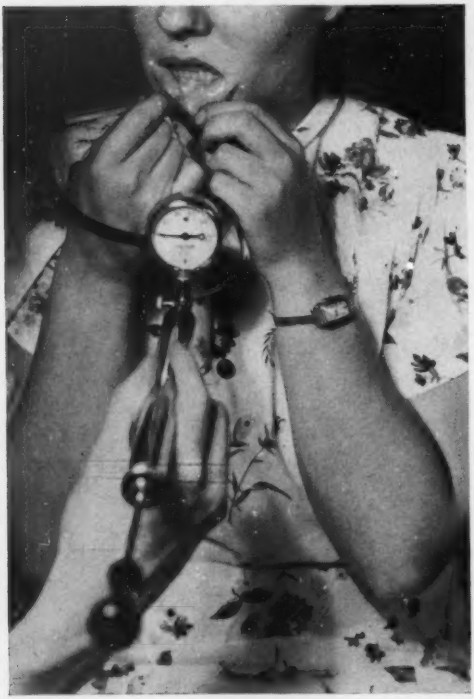

FIG. 6

Starting with the premise of the likelihood of chronic deficiency in vitamin C manifesting itself by bleeding from the gums, this was sought as the primary subjective symptom, and when found associated with a history of dietary deficiency and not wholly explainable on the basis of local causes, led to the choice of a total of 55 patients who were subjected to the various capillary resistance tests. Their dietary histories showing deficiency in the better sources of vitamin C such as fresh fruits, vegetables, milk and milk products, together with a history of bleeding from the gums of long standing not entirely justified by local gingival conditions, indicated the possibility of vitamin C subnutrition.

Manifest scurvy was not present, nor could a diagnosis of subacute or latent scurvy be attempted from the clinical gingival condition. The most common type of bleeding was, by far, that which occurred on direct traumatization with the toothbrush or on chewing hard foods, and its localization was not, as a rule, difficult for the patient. A few, however, gave an obscure history of bleeding occurring during the night relatively spontaneous in nature since no known trauma was effected.

FINDINGS WITH THE CAPILLARY RESISTANCE TESTS

The results of the various capillary resistance tests indicate that diminished capillary strength in apparently healthy individuals may, if viewed critically, serve as an indication of vitamin C undernourishment. Individual variations are the rule rather than the exception, making a number of determinations essential for a view of the trend and the establishment of the individual normal under treatment; broad generalizations for each test are permissible in setting up ranges of normality. With the other influencing factors eliminated, capillary fragility may be said to represent a clinical scurvy or a "subclinical scorbutus." Those patients giving a history of a diet which was relatively good were in the main found to give lower readings.

1. *The Gothlin Test.* The series of patients under consideration showed a variation from a low of 0 petechiae at 50 mm. positive pressure with the sphygmomanometer to a high of 18 petechiae. Eight patients of the series showed a capillary fragility that was in the higher brackets and thus suggestive of a C-avitaminosis. Gingival bleeding was a subjective symptom in each case, but the severity of the symptom varied in that 3 gave a history of relatively spontaneous bleeding while the remainder indicated that only a mild but definite traumatization would bring it about.

2. *Resistometer on the arm.* The negative pressure procedure using the Da Silva-Mello apparatus as previously outlined was followed throughout. The readings in the series of patients were found to vary between 0 and 22 macroscopic petechiae with the 45 cm. constant negative pressure. Limits of normality have been set through correlation with both the dietary history and the result of the Gothlin test. The normal range of petechiae formation varies from 0 to 6. Between 6 and 10 petechiae there exists a range of intermediacy, while the production of a greater number than 10 petechiae would indicate a subnormal capillary resistance.

A number of important factors should be considered in the evaluation and interpretation of these determinations:

A. The cup of 1 cm. diameter may include varying numbers of capillaries in determinations on the same individual or in different individuals; that is, distribution of skin capillaries on the inner surface of the forearm is not constant.

B. Not only capillaries but also other minute skin vessels such as arterioles and venules are probably involved in the production of macroscopic petechiae[8].

C. Thickness and resistance of the cutaneous tissues to distention vary in adjacent skin areas on the same individual and in different individuals.

This last factor mentioned, which may be considered mechanical in nature, may, as the sole variable, cause rather wide variation in the numbers of petechiae which will be formed by the constant negative pressure. A seeming lack of correlation occurred in a small number of cases between the positive and negative methods of determination; the Gothlin test would indicate a normal state of capillary resistance but the Resistometer test would give a reading in the higher brackets. This phenomenon, it is significant to note, occurred with individuals of rather debilitated state and sallow complexion, who, while their diet would not be seriously deficient, still gave a history of bruising easily and of readily getting the formation of so-called "black and blue spots" on minor trauma. It can therefore be seen that a small variation in the number of petechiae formation in an individual is of relatively little significance. Only changes of considerable magnitude would be indicative of significant modification in the intrinsic resistance of the capillary wall.

3. *Resistometer on the gingiva.* Local conditions of inflammation, edema, hypertrophy, etc., definitely obscure the formation of petechiae, and tend to complicate the results. The interproximal papillae of the upper and lower anterior teeth best lend themselves to the examination and were used throughout. Efforts were made, wherever possible, to include relatively normal appearing papillae, or papillae typical of the mouth, rather than only those showing pathological changes. Since the negative pressure exerted where pathology exists must act through a greater than average depth of material before affecting the capillaries, it will then be seen that the normal papilla will not only show a range of petechial formation tending to be greater in all cases, but should also vary in number with the strength of the capillary wall.

The results in the series of patients under consideration indicate the justification for this reasoning. The lack of formation of petechiae which occurred in areas where the papilla had undergone change due entirely to local causes (such as the irritating effect of calculus, etc.), together with the formation of petechiae in the same mouth on a relatively normal appearing papilla under the standard conditions, were both significant.

4. *Resistometer on the inner surface of the lower lip.* In using this apparatus on the tongue side of the inner surface of the lower lip, care must be exercised in localizing the 2 mm. area on which the negative pressure is to be exerted. Two or more areas are tested to act as checks on one another. On compression with glass the tested area shows a line of demarcation as a border. Strong light is used and the area compared before and after the application of the pressure.

The range of petechiae formation in the series under consideration varied from 0 to 3, with an undoubted tendency for the greater number to occur in those patients who showed capillary weakness elsewhere with the other tests used. The obvious necessity for using a narrow orifice of 2 mm. in the glass applicator is of course instrumental in leading to the narrow range, and the weaknesses previously outlined and the variations to be considered in using capillary resistance testing on the arm with the modified Dalldorf method hold true here. Because of the histological structure of the inner surface of the lower lip, with its macroscopically visible small vessels and its thin overlying mucous membrane, together with the result indicating variations in capillary resistance occurring in the region, lead to its recommendation as a site for further investigation.

Correlations and conclusions as to the value of the resistometer tests on the gingival tissues and mucous membranes can be attempted only with a large number of determinations. It may be noticed that the greatest percentage of patients giving a history of gingival bleeding showed, in spite of possible and questionable diet lacks, normal capillary resistance and normal blood pictures, indicating that the factors of local changes affecting the gingival tissues, uncomplicated by systemic disturbances, are most frequently met with.

CLINICAL CASES OF CHRONIC DEFICIENCY

CLINICAL FINDINGS

Choice of Cases

From the group of patients subjected to the various examinations previously outlined, a total of five cases was found to give readings in the higher brackets with the capillary resistance tests, dietary historics showing deficiencies in vitamin C intake, and giving a history of bleeding from the gums. The study led to the investigation of the clinical appearance of the gingival tissues and their adenexae, the clinical syndrome of the specific deficiency, the vitamin C excretion rate and the various changes which occur with periodontal treatment and the institution of vitamin therapy. Only those patients were chosen who showed absence of other systemic disorders which would tend to obscure and complicate the results.

Methods Used

1. *History.* A systemic history, as complete as possible was entered into to indicate the significance of such factors as endurance, rest, appetite, resistance to infection[20], weight changes, etc. Previous illnesses were recorded and, where indicated, the family physician was contacted for additional information. An oral history was taken to show the care and oral hygiene ordinarily practiced by the patient and the dental treatment previously received. The diet was determined in both its wide general tendencies and its specific content for repeated weekly periods. The history was completed with the recording of both the subjective and objective oral symptomatology.

2. *Blood examination and urinalysis.* Periodic investigation of this nature was undertaken to both eliminate the possibilities of blood dyscrasias and kidney dysfunctions that would effect the capillary resistance, and to establish and verify the blood picture in cases of chronic vitamin C deficiency[21] and the blood changes occurring with vitamin therapy[22].

3. *Capillary resistance testing.* The various capillary resistance tests previously outlined were employed periodically to determine the changes in the small vessels insofar as their tendency to maintain their integrity was concerned.

4. *Vitamin C excretion rate.* Both the micro method of Birch as described by Harris and Ray[23] and the method of Emmerie and Von Eekelen[24] were experimented with for the determination of cevitamic acid excretion in urine. They and others[25, 26] were found to be unsatisfactory for our

purposes and the more direct titration method following was used throughout in these cases. Twenty-four hour samples of urine were collected in dark brown bottles[37] containing glacial acetic acid as a preservative[38]. The 2:6 dichlorphenolindophenol dye solution was standardized against a solution of pure cevitamic acid equivalent to .025 mg. One cubic centimeter of the urine-acetic acid mixture as collected in the twenty-four hour sample was then titrated with the standardized dye solution[38], and calculations made on this basis. Determinations were made in two parts, that is, on twelve hour specimens.

The normal excretion rate[40 41] was taken to vary from 30-33 mg. per twenty-four hour sample of urine. When the findings were markedly below this rate it was taken as strong evidence of deficiency, and conversely, rates in this range or above were indicative of lack of deficiency or recovery.

The blood level of vitamin C was not entered into since a number of investigators[42 43] have shown a lack of relationship to deficiency and vitamin feeding.

5. *Capillary microscopy.* The capillary microscope known as the Ultropak was employed in these cases in making observations as to the number, structure, and relative tone of the small vessels of the gingival tissues and the inner surface of the lower lip in vivo. The normal was determined by study on cases without vitamin C deficiency who presented with periodontal structures which appeared normal clinically.

6. *Gingival biopsy.* Where possible, typical interproximal papillae of the gingiva were biopsied and sections prepared for study*. These were examined especially for their blood vessels and tissue structure both before and after treatment. For the sake of comparative investigation similar gingival biopsies were taken on other patients not showing vitamin C deficiency but clinical signs of definite periodontal disorders and giving the symptom of bleeding.

CLINICAL FINDINGS

The gingival picture in these cases of chronic deficiency in vitamin C was of such nature that only a tendency toward certain deviations can be described since no specific characteristics seemed to be common to all or distinctive from such gingival changes as occur with other periodontal disorders. In contrast to the usual finding in cases of manifest or frank scurvy where the gingiva are described as becoming blue and bloated, with the epithelium thin and of onion-skin appearance, together with a tendency to the formation of elevated pustule-like areas which exude a sero-purulent fluid on being ruptured[44], the gingiva in these cases of chronic deficiency showed a bluish-red discoloration, a bent toward a smooth glistening epithelium due to a slight edema, a thickening of the marginal gingiva, and a considerable loss of gingival tone.

Bleeding was patently a finding in all cases, with the history showing a pronounced tendency toward gingival bleeding of the spontaneous type where less injury would lead to the symptom than was ordinarily the case. No definite interference seemed to occur with the menstrual periods of the three female patients, nor did bleeding elsewhere in the body such as from the nose occur, with the exception of the tendency to petechiae or "black and blue spots" forming with slight trauma on the limbs on four of the five patients.

The patients as a group did not appear undernourished or emaciated. Their histories substantiated the work of previous investigators as to lack of resistance to infections such as the common cold, they tired easily, and suffered from anorexia to varying degrees. These would seem to be among the significant findings in the syndrome of chronic vitamin C deficiency.

COURSE OF TREATMENT

In common with most periodontal disorders, the single factor of the specific dietary deficiency was not the only cause of the gingival disturbances in any of these mouths. The rationale of treatment, therefore, could not be solely dependent on dietary change but had to take into consideration the necessity for the removal of all causes involved, the repair of the

*All sections prepared through the cooperation of Charles G. Darlington, M. D., and Gregory N. Brown, M. D., Department of Pathology, New York University College of Dentistry.

damage resulting from these various causes, and the raising of tissue resistance[46]. A regular course of conservative periodontal treatment was instituted including removal of visible deposits, curettage, balancing of occlusion, and proper toothbrushing procedure. Care was taken to place these patients on a balanced diet with a high vitamin content, to which was added 50 mg. a day[46,47] of cevitamic acid ("Cebione Merck"). Cevitamic acid by injection[45,48,50,51,52] was not given.

RESULTS WITH TREATMENT

Subsequent to the original determinations, active periodontal treatment and diet changes were instituted. This group of five patients showed a variation of from six weeks to four months as to the duration of such course of treatment, depending on the severity of the condition and the response to treatment.

1. *Blood examination and urinalysis.* The latter, used to indicate the absence of such systemic conditions as diabetes, nephritis, etc. did not, of course, vary. The first blood examination showed that three of the five patients suffered from a mild secondary anemia undoubtedly related to the malnutrition, with the platelet count, bleeding time and clotting time remaining, however, in the normal range. The remaining two patients showed no significant variations from the normal. With periodic determinations, including one at the conclusion of treatment, no definite increase in platelet count, or decrease in bleeding time or clotting time could be noted, though improvement in the blood cellular elements and hemoglobin occurred in those patients having the original secondary anemia.

2. *Capillary resistance testing.* Each patient in the group showed capillary fragility in the higher brackets with the standard tests previously outlined. The changes with treatment were noted, and the capillary resistance had returned to normal limits when treatment was concluded. The determinations on the gingival tissues and the inner surface of the lower lip were not conclusive evidence of such change due to the difficulties in their evaluation.

3. *Vitamin C excretion rate.* Considering the normal limits as 30-33 mg. per twenty-four hours, the excretion rate in the urine varied from 4.50 to 10.65 mg. in this series of patients at the beginning of treatment, and together with the other tests, was taken as strong evidence that a chronic deficiency did exist. With the diet change and the addition of 50 mg. of cevitamic acid "Cebione Merck" daily, the urinary excretion rate reached normal or higher than normal limits, and indicated excess after saturation of the tissues.

4. *Capillary microscopy.* Only tentative experimental efforts have thus far been made with this means of observing the gingival capillaries and the small vessels of the inner surface of the lower lip directly in the mouth. The cases of deficiency observed showed, however, in comparison with normal gingival tissues, a marked dilatation of the small vessels, a greater length of the vessels observable (probably related directly to the thinness of the overlying epithelium), and a seeming increase in the number of vessels present in analogous fields. Efforts will be made to get photographic evidence of these changes when technical problems involved can be solved[49].

5. *Gingival biopsy.* Significant changes were found to be present in pathological sections[54] taken at the interproximal papillae in these cases of chronic vitamin C deficiency. The outstanding variations from the normal found are: *a.* the thinness and dilatation of the capillary walls and the tendency to extravasation into the tissue proper, *b.* the connecting tissue collagen is markedly degenerated and granular rather than fibrillar.

Since bleeding frequently occurs associated with both simplex periodontitis (schmutzpyorroe), and with hypertrophy, sections of each were prepared to show their differences in contrast to two of the treated cases of chronic vitamin C deficiency.

A. *Schmutzpyorroe.* A characteristic case of heavy calculus deposition was biopsied, showing clinically a chronic inflammation and irritation of the gingiva, and a bluish coloration of the tissues related to a venous stasis. Bleeding occurred on trauma but no vitamin C deficiency was found. Sectioning from an area typical of the mouth showed (Fig. 7) that the

FIG. 7

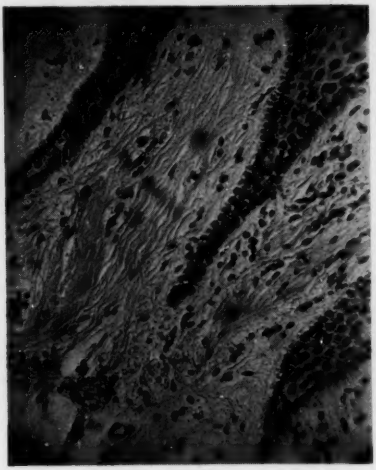

FIG. 8

epithelial pegs penetrate to varying depths. The papillae are uneven in height and appear as oval windows in a fenestrated epithelial membrane. The directional alignment of the fibers is normal. The underlying tissue (Fig. 8) generally is poorly vascular with the small vessels thickwalled and never dilated. Occasional lymphocytes streak the loose connective tissue planes.

B. *Hypertrophy.* A case of hypertrophy (Fig. 9), fibrous in nature occurring in a girl sixteen years of age and of three years duration. The etiology of the condition is probably related to mouth breathing. The tissues bled on slight trauma but no concomittant vitamin C deficiency was found. A typical section (Fig. 10) shows the epithelium to be unevenly hyperplastic, with the pegs long, pointed and often bifurcated. The epithelial cells flatten out toward the surface with their long axis parallel to it, and tend to become hydropic. Here, additional surface "keratin" takes the form of bloated cells stuffed with large droplets of pink staining hyaline-like material. In high power (Fig. 11) the subepithelial tissues are seen to be unevenly dense and unevenly vascular. The collagen is fibrillar and shows no marked change to granular degeneration. In some fields, the tiny serving capillaries were found numerous in number but deep seat-

FIG. 9

FIG. 10

ed, and the blood cells in all vessels appear normal. The capillary walls are not overdilated. The tissue throughout is flecked with chronic inflammatory cells i. e. lymphocytes.

C. *Chronic vitamin C deficiency.* A biopsy was taken from a typical gingival papilla in a case where a definite vitamin C subnutrition was found in the patient, a female adult (Fig. 12). Examination of the section (Fig. 13) indicates that the surface and subsurface cells often show hydropic changes with the epithelium unevenly thickened and thinned. The epiderm generally has lost its characteristic form and ramifies into the underlying connective tissue; where it is thin the pegs are short, pointed, and curved. Papillae which carry tiny capillaries stuffed with blood here reach to the surface. Where the epithelium is thickened in nature, the pegs are sickle-shaped or round and reach unevenly into the corium where the immediate subjacent tissue is flooded with cells. In place of normal connective tissue collagen (Fig. 14), the stroma surrounding these cell masses is not fibrillar but granular and degenerated. The predominating cell is the lymphocyte. Small serving vessels are rare and several are widely dilated, but the contents of these vessels are normal in appearance. The collagen tends to condense into twisted bundles in the depths of the section; even here, however, losing its intense eosin stain and being granular and degenerated.

D. *Chronic vitamin C deficiency.* Figure 15 shows a periodontal case of an

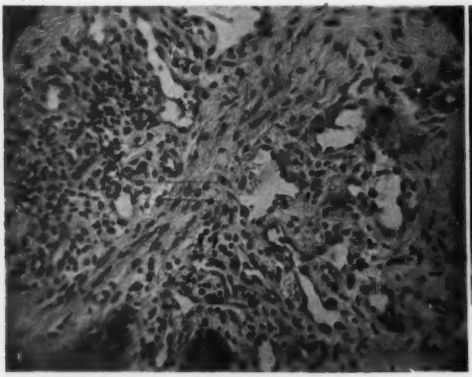

FIG. 11

FIG. 12

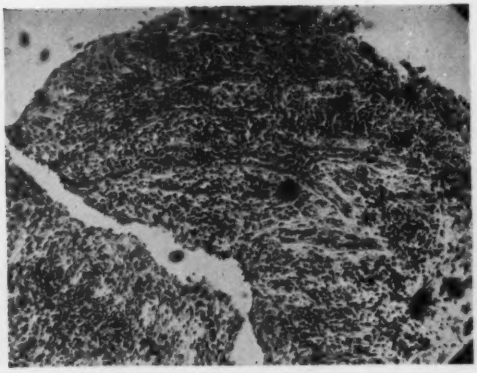

FIG. 13

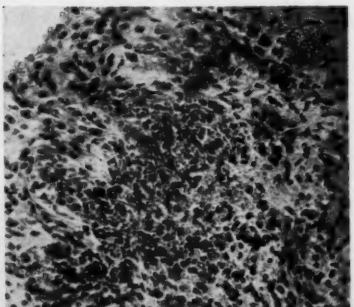

FIG. 14

FIG. 15

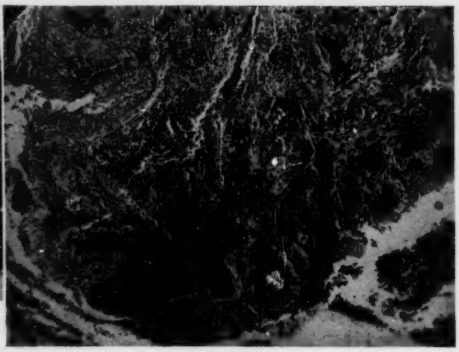

FIG. 16

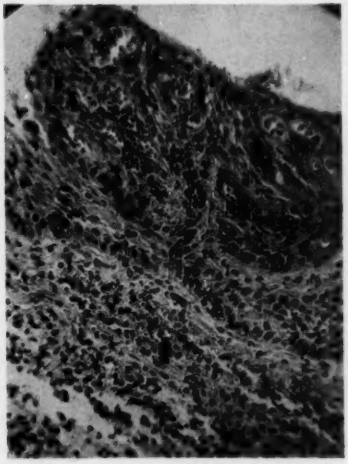

FIG. 17

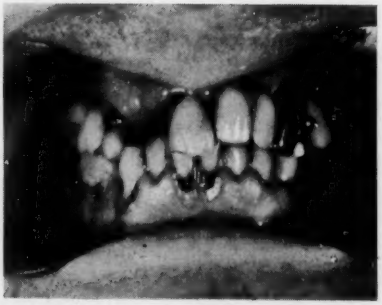

FIG. 18

adult male where chronic vitamin C deficiency was found to be associated. Radiographically all the upper teeth were found to be hopeless and were condemned but treatment was instituted for the lower anterior teeth. A section typical of the mouth (Fig. 16) shows the epithelium sending pointed pegs deep into the tissue beneath. It is sometimes difficult to trace the outline of the epithelium pegs and the basement membrane. The papillae can be seen to penetrate unevenly into these tissue masses (Fig. 17) so that the tiny serving vessels reach almost to

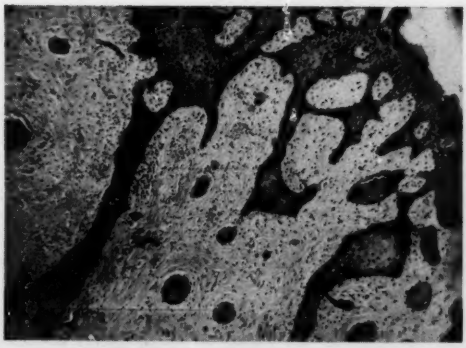

FIG. 19

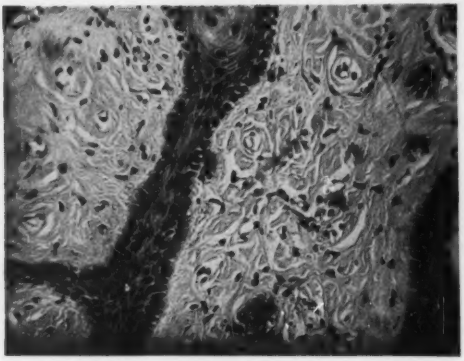

FIG. 20

the surface and are frequently dilated. Several have ruptured and blood has escaped into the surrounding tissue. The supporting stroma is flooded with chronic inflammatory cells, while below this reaction the collagen is in poor condition. It is no longer fibrillar but granular, "moth-eaten," and degenerated.

E. *Chronic vitamin C deficiency after treatment.* The previous case was treated by conservative methods and vitamin therapy instituted (Fig. 18). There no longer was any systemic evidence of deficiency and the gingival tissues have returned to a healthy state. Sectioning of a typical gingival papilla (Fig. 19) shows an epithelium which ramifies widely in the underlying tissues. The papillae, as a result, appear as islands enclosed by epithelial masses and their collagen is fibrillar. The basement membranes are preserved. In high power (Fig. 20) the tiny serving vessels are never seen to be dilated. There is no extravasation of blood into the tissues. The deeper collagen is normal and laid down in bundles and whorls. Inflammatory cells, whether single or clumped, are rare.

SUMMARY OF CONCLUSIONS

1—Bleeding from the gingival tissues is a widespread finding, and is related solely to the various local causes such as calculus deposition and insufficient and incorrect mouth hygiene in approximately 95 percent of all cases.

2—The Gothlin test and the Resistometer method for testing capillary resistance give results which can be correlated, and if carefully interpreted, act as an index of vitamin C insufficiency.

3—Urinary excretion estimation of vitamin C with 2:6 dichlorphenolindophenol dye is an accurate means of determining if a state of deficiency exists.

4—Latent or chronic deficiencies in vitamin C are met with, and manifest themselves in the soft tissues of the mouth.

5—Studies with capillary microscopy indicate that, in vivo, chronic deficiencies in vitamin C are related to a dilatation of the small vessels in the gingival tissues.

6—Microscopic study of gingival sections taken in cases of established chronic deficiency shows changes in the small vessels and in the tissues that tend to be characteristic, and differ from both the normal picture and those changes due to a simple chronic irritation.

7—No dimunition of specific blood element was found to be associated with the cases of chronic scurvy investigated, nor was any significant and characteristic change in the blood picture found to occur with institution of vitamin C therapy.

8—With institution of proper therapy in cases of chronic vitamin C deficiency, both local and systemic changes occur as indicated by increased capillary resistance and excretion rate, and changes in the gingival tissues as manifested in section and by capillary microscopy; bleeding from the gingival tissues is no longer a finding.

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THE TWENTY-FIFTH ANNUAL MEETING *of the American Academy of Periodontology will be held in Saint Louis, Missouri, October* 20, 21 *and* 22, 1938 *at the Coronado Hotel. Members of the American Dental Association are urged to attend.*

Salivary Calculus*

By THEODOR ROSEBURY, D. D. S., New York City[1]

Research on salivary calculus—on its nature, origin, manner of formation and deposition, and on the conditions which determine its appearance in individual mouths—has progressed to a point at which a statement of the problem as a whole may be of value as a base line for further investigation. It now appears clear that the several types of calculus, whether supragingival, subgingival, or duct calculi proper, are all similar chemically (Kranz, 1919), histologically (Naeslund, 1926) and bacteriologically (Naeslund, 1926; Taylor, 1934; Bibby, 1935); and it is likely that they have a common origin in saliva, their physical differences being conditioned by their respective locations (Kronfeld, 1933). The inorganic fraction of calculus, comprising about 70 to 80 per cent of the mass, appears to consist chiefly of tricalcium phosphate, with smaller amounts of calcium carbonate and magnesium phosphate, and perhaps of calcium hydroxide (Karshan and Schroff, 1928). These substances may be present in the form of apatite salts similar to those of bone (Taylor and Sheard, 1929). The organic fraction, comprising about 15 to 25 percent, is of uncertain composition. It has not been studied chemically; but histological and bacteriological investigations suggest that it consists largely of the bodies of filamentous microörganisms, members of the ill-defined genera *Leptotrichia* or *Actinomyces*, arranged as an interlacing or radially disposed stroma, and viable even in the interior of the deposit (Bulleid, 1924; Naeslund, 1926; Taylor, 1934; Bibby, 1935). It is interesting to observe a general agreement in the data of several investigators on the inorganic composition of calculus of all three types, an agreement which, especially in view of differences in analytical methods employed, suggests that salivary calculus is not a haphazard deposit but the result of a consistent and uniform process involving a selection of certain salivary constituents (Schehevetsky; Talbot, 1913; Kranz, 1919; Prinz, 1921; Karshan and Schroff, 1928).

Deposits similar in physical properties to salivary calculus have been produced from stagnating saliva *in vitro* (Prinz, 1921; Naeslund, 1926; Hall and Westbay, 1925), from artificial mixtures of salts and protein made to resemble saliva (Prinz, 1921), and from culture media containing calcium salts and inoculated with pure cultures of filamentous bacteria (Bulleid, 1924; Naeslund, 1926; Bibby, 1935). Bacterial growth seems to have played an essential part in the formation of all such deposits, since their deposition has been inhibited in similar experiments by the addition of disinfectants, or in sterile media with the use of killed cultures (Naeslund, 1926; Bibby, 1935). Neither the pH of the medium nor loss of carbon dioxide seems to have been important in their formation. Yet the possibility remains to be thoroughly investigated that one part of the mechanism of the deposition of calculus depends upon the production of an alkaline reaction in the immediate environment of bacterial growth, rather than in the medium at large. Another possible agency, also worth additional study, is concerned with colloidal changes in saliva (Karlström and Miller, 1928); the formation of calculus may be induced or favored by the decomposition of colloids, perhaps itself induced by bacterial action.

The enzyme phosphatase, capable of liberating inorganic phosphate from organic combination, has been extracted both from human gum tissue (Adamson, 1929) and from desquamated epithelial cells in saliva (Smith, 1930), and has been shown capable of forming concretion soutside the mouth. Data relating to this enzyme, however, are difficult to reconcile with those dealing with bacterial action. Deposits of artificial calculus have certainly been produced in the absence of tissue enzyme; and

*Abstract of a paper read at a meeting of the American Academy of Periodontology, Atlantic City, N. J., July 10, 1937. Much of this material has been treated at greater length in a previous review by Rosebury and Karshan (Annals of Dentistry 3:37, 1936.)

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if the inference mentioned above is true—that a uniform mechanism operates in the deposition of calculus—the participation in the process of phosphatase derived from tissue cells seems doubtful.

Many of the theories advanced to explain the formation of calculus seem untenable in the light of information derived since they were advanced. The theory (Moore, 1910; Kirk, 1911) which regards loss of carbon dioxide from saliva as the essential condition for the precipitation of calculus is invalidated by the findings (a) that the change in carbon dioxide-content of saliva on exposure to air is actually insignificant (Clark and Carter, 1927), and (b) that limitation of the escape of carbon dioxide from stagnating saliva *in vitro* does not affect the formation of artificial deposits (Hall and Westbay, 1925; Naeslund, 1926). The theory of Prinz (1921), in which precipitation is considered to depend upon loss of protective colloid effect as a result of surface concentration of colloids in stagnating saliva, may be questioned in the light of more recently developed data on the action of filamentous bacteria, as well as on its basic premise: it seems hardly likely that the oral regions nearest the salivary gland ducts, in which calculus forms by preference, afford better conditions for stagnation of saliva than other regions of the mouth. Broderick's view (1936) that the development of salivary calculus depends upon a generalized alkalosis reflected in increased alkalinity of saliva is in conflict with evidence that the saliva, unlike the urine, is little affected by changes in the acid-base equilibrium of the blood (Smith, 1930; Champion, 1932), as well as with the finding noted above that the pH of the medium at large does not seem to be important in the formation of artificial calculus outside of the mouth (Hall and Westbay, 1925; Bibby, 1935). Investigation of the pH of saliva in relation to individual tendency to form calculus, to be mentioned below, has likewise provided little support for this theory.

The theory of Naeslund (1926) seems more consonant with the available data. According to this view certain filamentous microörganisms, which may be seen to constitute the matrix of naturally occurring calculus, can be isolated from it, and are capable of inducing the formation of similar deposits outside of the mouth, are assumed to induce the precipitation of calcium salts from saliva by causing a rise in pH in their immediate neighborhood, and by the decomposition of protein. These organisms, by growing as a compact vegetation firmly attached to the tooth surface, would then provide a framework to hold the deposited salts and secure the mass to the tooth. The preferential deposition of calculus in regions of the mouth nearest to the orifices of the salivary ducts would depend upon abundance of substrate rather than upon stagnation. The process would be progressive rather than self-limiting, since the organisms apparently remain alive within and at the periphery of the mass. Nevertheless the ability of the bacteria in question to induce both of the phenomena postulated in this concept —increased alkalinity and effective proteolysis—is in need of further verification.

An attempt has only recently been initiated to determine the underlying causes of variation in individual tendency to form salivary calculus by means of chemical studies of saliva correlated with such tendency. It appears that stimulated saliva of persons who form calculus in large amounts has, in average, more calcium and inorganic phosphate, and a higher carbon-dioxide-capacity, than that of persons with little or no such tendency. Differences in pH of saliva and in concentration of salivary protein, although in the direction to be expected from theoretical considerations, are too small to be considered significant in the groups thus far studied (Tenenbaum and Karshan, 1937). Additional evidence is available of a relative antagonism between the tendency of individuals to form calculus and the activity of dental caries in their mouths, on both clinical and analytical grounds, as derived from a study of Alaskan Eskimos (Rosebury, 1937; Karshan and Rosebury, 1937). An inverse correlation was observed between the occurrence of calculus and the prevalence of caries, apparently independent of age; and the saliva of persons who tend to form large deposits of calculus was

found to be chemically similar to that of persons free from caries (in group average characteristics), and *vice versa*. The occurrence of calculus has also been associated with a tendency of saliva to deposit cellular débris, and with poor oral hygiene. (Willsmore, 1937.)

Further research particularly on the chemical and bacteriological aspects of this problem may be expected to increase our knowledge of the subject materially, and seems warranted by its probable importance as an avenue of approach to the problems of gingivitis and periodontal disease.

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Salivary Analysis of Patients Subject to Wastings: Abrasions and Erosions*

By FRANCES KRASNOW, PH. D.; EDITH B. OBLATT, A. B., Guggenheim *Dental Clinic;* and ISADOR HIRSCHFELD, D. D. S., New York City

This report represents a preliminary study of a concerted effort to find laboratory criteria characteristic of different dental conditions. Although the greater number of erosion cases are patients of Dr. Hirschfeld with whom the work was originally started, the biochemical findings to be summarized presently are based also on examinations of patients of Drs. L. H. Berk, Freda Sale Burk, James M. Dunning, S. J. Fried, George D. Oblatt, Murray Raybin, Alfred Walker, Louis Wolf, the Guggenheim Dental Clinic, and the Dental Department of the Metropolitan Life Insurance Company.

All subjects were placed on a definite physiological plane for at least one week preceding any analysis. The requirements set were:

(1) No mishap during this week.
(2) Eight hours sleep the night prior to each test day.
(3) No brushing of the teeth or rinsing of the mouth, nor having breakfast, water, or smoking on the morning of each test.
(4) A one and one-half to two hour interval between rising and collections, this interval including a fifteen minute rest period at the laboratory.

Time does not permit to detail either the collection procedure[1] or the methods

*Read before the American Academy of Periodontology, July 9, 1937 at Atlantic City, N. J.

for the quantitative determination of the various salivary constituents[2,3]. Suffice it to say, that all are standardized techniques and have been employed for several years.

Chart 1 lists the averages obtained on total unstimulated saliva. A comparison of the figures reveals that the averages obtained for hydrogen ion concentration, calcium, magnesium, inorganic phosphate (P), protein, lipid phosphorus and cholesterol lie entirely beyond the averages for the same constituents in saliva of normal individuals. It may be noted that "normal" defines only subjects who, as far as medico-dental clinical and laboratory tests can discern, have no ailments. Also, for the most part, individuals included in the erosion and caries groups were not suffering from any other pathological involvement.

In tabulating the results our attention was drawn to the difference in the salivary picture between the cases with alkaline saliva and those with acid saliva. Classified as separate groups, the two types show characteristically different averages. The alkaline set indicates markedly lower concentrations: pH', calcium', magnesium and protein approach the normal; inorganic phosphate and cholesterol fall below, lipid phosphorus above. The acid subgroup is removed farther from the normal. Thus witness, pH, calcium, magnesium, inorganic phosphate, protein, lipid phosphorus and cholesterol with values 6.73, 8.2, 0.84, 19.7, 489, 0.60, 12.9 respectively instead of 6.83, 7.6, 0.75, 18.0, 446, 0.52, 11.2.

For comparison a series of caries-active individuals has been similarly treated. Variations from the normal are fewer—non-overlapping figures appearing only for pH, magnesium, lipid phosphorus and cholesterol. Again, the differences are enhanced in the averages for the acid saliva subgroup, being changed to 6.71, 0.73, 0.22, 11.9 from the corresponding general averages 6.92, 0.65, 0.20, 10.7. For the alkaline saliva subgroup inorganic phosphate is reduced; pH, calcium and protein

CHART 1
SUMMARY OF ANALYSES ON TOTAL UNSTIMULATED SALIVA
AVERAGE VALUES WITH STANDARD DEVIATIONS
Expressed in Mg per 100 cc

Type of Case	Number of Cases	pH	Calcium	Magnesium	Phosphorus	Protein	Lipid Phosphorus	Cholesterol
Normal*	12	7.14±0.04	6.3±0.4	0.46±0.07	15.2±1.0	298±17	0.13±0.02	7.5±0.7
Erosion**	19	6.83±0.05	7.6±0.5	0.75±0.10	18.0±1.4	446±46	0.52±0.11	11.2±2.0
Erosion Alkaline saliva	4	7.19±0.07	5.5±0.5	0.42±0.04	12.2±1.6	284±33	0.26±0.11	5.3±2.1
Erosion Acid saliva	15	6.73±0.04	8.2±0.5	0.84±0.12	19.7±1.5	489±53	0.60±0.14	12.9±2.3
Caries***	24	6.92±0.06	6.2±0.2	0.65±0.06	14.4±0.6	346±24	0.20±0.03	10.7±0.77
Caries Alkaline saliva	9	7.21±0.04	6.0±0.25	0.63±0.08	12.6±0.7	315±33	0.19±0.02	9.2±0.7
Caries Acid saliva	12	6.71±0.04	6.4±0.19	0.73±0.08	15.2±0.7	359±39	0.22±0.04	11.9±1.2

*Normal refers to subjects who as far as medico-dental clinical and laboratory examinations can discern have no ailments. The age range, with few exceptions, does not exceed 10 years.

**Erosion refers to subjects suffering from wastings: erosions or abrasions.
***Caries refers to subjects with active decay processes only.

CHART 2
PERCENTAGE DISTRIBUTION OF VALUES FOR ANALYSES ON TOTAL UNSTIMULATED SALIVA
WITH REFERENCE TO THE AVERAGE NORMAL RANGE

Type of Case	Hydrogen ion Concentration Below	Above	Calcium Below	Above	Magnesium Below	Above	Phosphorus Below	Above	Protein Below	Above	Lipid Phosphorus Below	Above	Cholesterol Below	Above
Erosion	11	89	21	74	16	68	32	53	21	68	11	78	33	57
Erosion Alkaline saliva	—	—	75	25	50	0	75	25	75	25	50	50	75	25
Erosion Acid Saliva	—	—	7	87	7	87	20	60	7	80	7	86	24	65
Caries	24	62	43	22	22	65	52	39	29	54	11	63	14	82
Caries Alkaline saliva	—	—	44	11	11	67	89	11	44	33	22	78	22	67
Caries Acid saliva	—	—	17	33	17	75	33	50	25	67	10	50	8	92

remain but magnesium, lipid phosphorus and cholesterol are high.

If, instead of considering averages, the percentage distribution with respect to the average normal range be tabulated, Chart 2 results.

In erosion, all constituents show a large per cent of the cases falling beyond the high normal value and this percentage is raised for the subclass with acid saliva. In caries, the same four constituents—hydrogen ion concentration, magnesium, lipid phosphorus, cholesterol—which were previously stressed as having non-overlapping averages, here present very high incidence of cases above the high normal level. Moreover, this incidence is accentuated for the caries-acid saliva subgroup.

To test further whether these differences are significant, computations were made for odds against their random occurrence. In chart 3, odds of three to one may be significant, four or more to one *are* significant[3]. Thus:

(1) Deviations from the normal pertaining to the erosion-acid saliva series are probably real for pH, calcium, magnesium, inorganic phosphate, protein, lipid phosphorus and cholesterol.

(2) Differences from the normal found for the caries-acid saliva group are probably real for pH, magnesium, lipid phosphorus and cholesterol.

(3) The calculations for the erosion-alkaline subgroup show no significant odds against randomness; those for the

CHART 3

ODDS AGAINST RANDOM OCCURRENCE OF DEVIATIONS
CALCULATIONS BASED ON D/P. E.
ANALYSES ON TOTAL UNSTIMULATED SALIVA

Compared Groups	pH	Calcium	Magnesium	Phosphorus	Protein	Lipid Phosphorus	Cholesterol
Normal—Erosion	1341	6.3	8.5	2.6	22.2	37.4	3.5
Normal—Erosion, alkaline saliva	1.0	1.6	1.0	2.6	—	1.4	1.0
Normal—Erosion, acid saliva	14,700,000	13.6	13.6	9.9	44.8	44.8	7.3
Erosion, alkaline saliva—Erosion, acid saliva	1.0	142.3	31.4	64.9	37.4	4.6	9.9
Normal—Caries	26.4	—	5.4	1.0	3.0	7.3	31.4
Normal—Caries, alkaline saliva	1.9	1.0	2.2	6.3	1.0	4.6	3.0
Normal—Caries, acid saliva	427,000	—	9.9	—	2.2	3.5	26.4
Caries, alkaline saliva—Caries, acid saliva	14,700,000	1.6	1.0	11.6	1.0	1.0	8.5

"—" means D/P.E. is less than 0.5.

CHART 4

ANALYSES ON TOTAL UNSTIMULATED SALIVA
SUMMARY OF RESULTS OBTAINED BY THREE COMMON STATISTICAL METHODS FOR EVALUATING PROBABLE SIGNIFICANCE OF FIGURES

Analysis	Erosion									Caries					
	Average Range Not Overlapping Normal			Distribution More Than 50% Above Average Normal Range			Odds Against Random Deviations			Average Range Not Overlapping Normal			Distribution More Than 50% Above Average Normal Range			Odds Against Random Deviations		
	Total	Alk.	Acid	Total	Alk.	Acid	Total	Alk.	Acid	Total	Alk.	Acid	Total	Alk.	Acid	Total	Alk.	Acid
H+	√		√	√	√		√	√		√		√	√		√	√		√
Ca	√		√	√	√		√						√					
Mg	√		√	√	√		√	√		√	√?	√	√	√	√	√		√
P	√	√	√	√	√		√?	√			√						√	
Pr	√		√	√	√		√	√			√?		√		√	√?		
L. P.	√		√	√	√		√	√		√	√	√	√	√		√	√	√
Chol.	√		√	√	√		√	√		√	√	√	√	√	√	√	√?	√

caries-alkaline subgroup resulted in odds of three or above for inorganic phosphate, lipid phosphorus and cholesterol.

There is apparently a very close agreement of the three methods for evaluating probable significance of the analytical figures differentiating the salivary composition accompanying abnormal dental conditions from the normal chemical make-up of saliva. This relationship is graphically summarized in Chart 4. Take for example the erosion-acid saliva analyses. It appears first, that none of the averages overlap, hence variations from the normal are probably significant. Then, the incidence of higher than high normal values is above 50 per cent for all constituents—thus again pointing to the probable significance of the results. And, finally the odds against random occurrence of differences are above four for all substances studied and so the suggested probability is verified a third time. The data for the other groups follow a similar regularity.

It seems plausible to assume, therefore, the likelihood that

(1) Individuals suffering from erosion with accompanying acid saliva show specifically characteristic high levels for salivary calcium, magnesium, inorganic phosphate, protein, lipid phosphorus and cholesterol. Erosion with alkaline saliva tends to low or normal values.

(2) The chemical composition peculiar to caries associated with acid saliva is characterized by high magnesium, lipid phosphorus and cholesterol. Caries with alkaline saliva shows in addition low inorganic phosphates.

How applicable these findings may be is of course difficult to predict. It has been possible, however, to frequently match successfully the classification set up on the basis of clinical criteria. Thus, cases in which the erosion process has attacked the enamel and cementum and worn away dentine possess an acid saliva with high cholesterol.

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1—Krasnow, F., Oblatt, E. B. and Kaplan, N. Journal of Dental Research 15:367, 1936.

2—PH determination: A series of standards in glass cells are prepared by adding to 0.05 cc. indicator (generally brom thymol blue) 0.2 cc. buffer mixtures containing graded concentrations of M/15/Na_2HPO_4 and M/15/KH_2PO_4. Comparison with these gives the pH of the unknown (0.05 cc. indicator, 0.15 cc. water adjusted to pH 6.7-7.0, and 0.05 cc. saliva) in a similar cell. This procedure is usually checked with that outlined in the Journal of Dental Research 11:573, 1931.

3—Krasnow, F., Karshan, M. and Krejci, L. E. Journal of Laboratory and Clinical Medicine 17:1148, 1932.

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8—Pearl, R. Introduction to *Medical Biometry and Statistics*. Second Edition, 1930.

"Knowledge and timber should not be much used until they are seasoned."

Factors Influencing the Kinds and Numbers of Mouth Bacteria*

By J. L. T. APPLETON, B. S., D. D. S., SC.D.
Philadelphia, Pa.

SINCE LEEWENHOEK, bacteria have been known to exist in the human mouth in large numbers. Pasteur and Sternberg independently discovered the pneumococcus in saliva, and in Loeffler's first description of the diphtheria bacillus he announced the isolation of this micro-organism from the mouth of a healthy subject. Since then many kinds of bacteria have been found in the human mouth; including the tubercle bacillus, the typhoid bacillus, the spirochete of syphilis, the tetanus bacillus, the "influenza" bacillus, the various pus-producing germs, etc.

The first systematic studies of the oral flora and its significance in local and systemic disease were made by W. D. Miller in the last 2 decades of the 19th century. Goadby in England to some extent carried on and supplemented Miller's work. Since then there has grown up a large literature dealing with this field. As a result we have learned that the oral flora changes from birth to old age; but that for a given individual this flora is relatively characteristic and constant, both as to the kinds of bacteria and as to their numbers.

We have learned enough to regard the above statements as being in general true: and we have learned enough to regard certain kinds of bacteria as being, to say the least, suspiciously related to dental decay, to some of the diseases of the dental pulp, of the gums and of the supporting tissues of the teeth in the jaws. We have also learned to suspect strongly that bacteria from the mouth may be aspirated into the respiratory tract or may be swallowed, and as a result may initiate disease or aggravate already existing disease in the respiratory tract or in the gastro-intestinal canal. Furthermore there is evidence that at least occasionally bacteria which have first gained a foothold in the dental pulp or in the supporting tissues of the teeth, may be carried through blood or lymph to remote parts of the body and may in these remote parts set up or aggravate disease.

Thus from a standpoint of health and disease we have learned that mouth bacteria may be related not only to mouth diseases but also to disease elsewhere in the body and to general disease.

While we have learned all this, we have, unfortunately, learned very little of the factors which influence the kinds and numbers of mouth bacteria: and a knowledge of these factors lies at the base of a rational hygiene of the mouth and of the general hygiene of the body in so far as mouth conditions directly or indirectly effect the general health.

Oriented by the viewpoint outlined in the above paragraphs, the Department of Bacteriopathology of the School of Dentistry of the University of Pennsylvania began a few years ago a systematic, comprehensive and sustained attempt to recognize and to evaluate the factors which influence or determine the kinds and numbers of mouth bacteria. This attempt has been prosecuted ever since, in so far as time and resources have permitted, as a problem in pure biology. For the time being, at any rate, we are not thinking of any immediate clinical significance or application. That will be welcome if it comes, but while working such motivation had best be left out of account or relegated to a decidely secondary position.

The problem is stated above in its most general form and cannot of course be attacked in its entirety. It may, and must, be broken up into a host of subsidiary problems, each of which holds out hope of solution within a reasonable time. We do not feel justified in offering in detail a *complete* program of research in this field. It is not unlikely that the solutions of subsidiary problems, as they are made, will deepen and widen our understanding of the general problem, and will modify our program. The results of the study of some

* Read before the American Academy of Periodontology, July 9, 1937 at Atlantic City, N. J.

of these subsidiary problems are summarized below.

Survival of artificially introduced saprophytes in mouth.

I. Influence of number introduced.

Mouth rinsed with 5cc. suspensions of *Serratia marcescens* of various concentrations. After various periods of time (½ to 14 hours) during which nothing was taken into mouth, mouth again rinsed with 2cc. of diluent and attempts made to recover the bacterium from this second rinse. Work done on 3 individuals. Relation between density of suspension and length of survival in mouth, positive; and apparently varies with different individuals and in the same individual on different days. A detailed report of this work is now in preparation for publication in Am. J. Hyg., Sept., 1938.

In vitro effect of saliva on bacteria.

Inocula of various densities of *Serratia marcescens, Escherichia coli, Eberthella typhosa, Ps. aeruginosa seeded* into human saliva, incubated at 37°C., samples of these mixtures taken at various times (1½, 3, 6, 9 up to 24 hrs.). Relative number of these bacteria estimated in these samples. Saliva furnished by different individuals, centrifuged, in some instances sterilized by filtration, in some instances heated 56°C. 30 minutes, in some instances bacteria removed by centrifuging replaced after filtration or heating. In all instances centrifuged saliva antibacterial. Filtered or heated, centrifuged saliva less antibacterial. Antibacterial effect apparently restored by restoring bacteria separated by centrifuging.

Effect of oral mucosa on bacteria applied to it.

Suspensions of *Escherichia coli* painted on areas of oral mucosa (lingual dorsum, hard palate, cheek, mucous surface of upper lip, external alveolar plate of maxillary anterior teeth). Swabbings, taken immediately and after 3 min. (from symmetrical area), transferred to surface of plates containing steril gentian-violet agar. After 24 hrs. at 37°C. number of colonies from the immediate and the 3 minute swabbing compared. Results confirmed work of Arnold *et al.* that oral mucosa to some extent possesses a bacteria fixing or anti-bacterial effect analogous to the effect of the skin. The various areas of the oral mucosa varied in the frequency with which they exhibited this effect. A smaller series substituting *Serratia marcescens* for *E. coli* yielded similar results.

Mouse-protection tests with saliva.

Following principle of mouse-protection tests with antipneumococcic sera, attempt was made to determine if human centrifuged saliva exerted similar effect. Data obtained with unstimulated saliva of one individual. Similar, but fewer, tests with salivas of other individuals gave corroboratory results. Bacterium used: *Serratia marcescens.* Three sets of mice: (a) received intraperitoneally 0.5cc. of centrifuged saliva alone—only 1 death occurred in this group; (b) received intraperitoneally 0.5cc. of 24-hour broth-culture of bacterium, diluted 1:7 with steril broth; (c) received intraperitoneally 0.5cc. of 24-hour broth-culture of bacterium, diluted 1:7 with centrifuged saliva. After injection, animals (more than 200) observed for 72 hours. Deaths and their order noted. Heart-blood cultures taken from animals that died; only 2 or 3 failed to yield bacterium injected—these discarded in calculating results. After 72 hours, all survivors chloroformed and heart-blood cultures taken. There were (a) lower gross mortality, (b) postponement of death, and (c) fewer positive, heart-blood cultures among survivors, in mice receiving saliva suspensions than in those receiving broth suspensions. This work in progress with other bacteria, including pneumococcus, in place of S. *marcescens.* For detailed account see Zt. f. Stomat., 35:18-24. Jan., 1937.

4001 Spruce St.

Coöperation of the General Practitioner, Periodontist, and Patient*

By JUSTIN D. TOWNER, D. D. S., Memphis, Tennessee

Dentistry, even in its most restricted conception, implies the health conservation of the greatest number of teeth for the longest possible time. In its true and higher professional status it must assume full responsibility for the care of the mouth. "This includes not only the care of the teeth and their supporting tissues, the various forms of stomatitis of both local and systemic origin, the mouth lesions of generalized skin disease, the recognition of carcinoma and other malignancies, and the severe infections of mouth origin, whether they are ascending or descending in character." The incontrovertible fact is that no result other than clean, vital teeth in anatomic form and relation, with preservative restorations, and supported by healthy investing tissues, is good dentistry.

There is meager excuse for the development of periodontal disease of local origin in patients who visit the dentist at frequent intervals. Its presence is definite evidence of either inadequate hygienic care, indifferent coöperation, poor supporting tissue resistance, or neglect on the part of the dentist. Usually two or more of these influences conspire to the detriment of the periodontium until nature, in extremity, cries out for relief. Then, it often develops that the disease is sufficiently advanced to preclude saving otherwise healthy teeth.

Periodontia, as a specialty, is chiefly the result of neglect of this important practice by the general dentist. Even so, the periodontist is in no sense independent since, by reason of the interdependence of all dentistry, he must ultimately rely upon a professional confrere to protect and make more secure any favorable results of his services. The principle hope of successfully controlling these lesions, as they present, or of staying the tide of their ever increasing prevalence, lies in intelligent and concerted effort. In this respect there is a trinity interest:—that of the general practitioner, the periodontist, and the patient. It matters not into whose practice a case may come, the triad is essentially the same.

Since ninety percent of adults are the hosts of some form of periodontoclasia and only approximately ten percent receive treatment, other than extraction, directed either toward its prevention or cure, the coöperation of the general practitioner becomes imperative in any attempted control of these diseases.

It is eminently fair to expect all dentists to discover periodontal lesions, regardless of their disposition in respect to treatment. Though relatively few readily adapt themselves to the exacting demands of this phase of modern practice, the profession generally cannot escape the responsibility of at least competent examination, intelligent diagnosis, and reference of cases. This elementary evidence of coöperation with the periodontist in the interest of the patient, while significant and important, is by no means sufficient.

The general practitioner should, first of all, base his practice upon the health of supporting structures as fundamental to all rational professional service. If these are diseased, even incipiently, their claim upon him, with the exception of emergencies, is primary. Protection of these tissues against detrimental influences and relief of causative factors in their distress should be his constant purpose. Accordingly, open contacts, whether due to caries, fracture, or drifting should be properly closed to prevent menace to the periodontium. Loose teeth indicate the necessity of either extraction, splinting, or relief of excessive stress. Edentulous spaces must be bridged to restore function, and to avoid shifting of remaining teeth. Restorations of various types demand accuracy in marginal fit, anatomical form, proximal contact, occlusal relation, and soft tissue adaptation. There is, too, the ever present necessity of educating the patient in approved

*Read before the American Academy of Periodontology, July 9, 1937 at Atlantic City, N. J.

personal mouth care. All this, and more, the general practitioner may do before, during, and after treatment by the periodontist of referred cases. In no more substantial way can coöperation be expressed.

The periodontist should be concerned primarily in defeating the ravages of the diseases he has elected to treat. He may manifest this disposition in his own specialized practice and by stimulating the profession to a better understanding of periodontal disease processes and their intimate relation to general dentistry.

Realizing that the general practitioner is usually the first to contact these conditions he should insist upon an early diagnosis by him when cure is easily and substantially accomplished. It is his duty to encourage and sustain the efforts of those who attempt to master the requisites, principles, and technique of periodontia.

In referred cases, showing poor dentistry, it becomes necessary to determine the ability of the former dentist to render adequate supporting services. If coöperation is weak in this respect it must be improved to a point of acceptability, else the periodontist is justified in either declining to share the responsibility of certain failure, or calling to his aid others more capable of contributing to success. An alternative is the establishment of group practice wherein all may work in close accord for a common good. In any circumstance, the patient's welfare is of chief importance and must be judiciously conserved regardless of hazard.

Ideal relations of the general practitioner and periodontist, with skillful expression of their respective services, prove disappointing without the whole hearted assistance of the patient, whose recovery is of foremost concern. It is this support that completes the tripod of coöperative effort.

The requisites of all recovery are: natural or acquired resistance to disease; coöperation of the patient in permitting good dentistry, maintaining proper oral hygiene, and observing rational laws of general health; and the character of professional service rendered. In this trinity the patient's responsibility is shown to be at least equal to, if not greater than, that of the dentist. The sooner such a truth is recognized and accepted by the professional and laity alike, the more beneficial and appreciated will be our efforts. Certainly, each individual should be aware of the definite obligation assumed.

Coöperation of the patient is evidenced in confidence, chair deportment, and personal mouth care.

Confidence, the basic principle of all favorable issues, is essentially a stimulating and controlling influence, without which there can be no satisfactory relations.

Chair deportment, the patient's behavior under stress, is largely accountable for success or failure. It is here that temperament manifests itself, which, if expressed adversely, demands treatment in addition to that given the disease. An antagonistic, even indifferent disposition, or inability and unwillingness to endure discomfort decidedly limits the amount and quality of dental services.

Personal mouth care contemplates intelligent and persistent effort in both a defensive and aggressive program of oral hygiene. The objects to be attained are the removal of predisposition to disease and destruction of its exciting causes. The degree of coöperation here depends appreciably upon instruction regarding the use and care of teeth and important adjacent tissues. With the significance of function properly impressed and approved technique of care thoroughly understood, the patient is in position to coöperate fully. If this is done acceptably and the support of the general practitioner is favorable, periodontia will be more interesting, definite, and mutually satisfactory.

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A Board of Periodontology

THE NEED for some means of examining and certifying the qualifications of those who have become experts in some one of the branches of dentistry is becoming as apparent in Dentistry as it has for some time in Medicine. Six branches of dentistry have become so specialized as to require this treatment. These are Orthodontia, Oral Surgery, Periodontology, Pedodontia, Prosthodontia, and Public Health Dentistry.

The Academy of Periodontology is taking the initiative in the organization of boards to formulate standards of preparation, examine candidates and certify to their proficiency in these various specialties. It is doing this by requesting all of the national specialty dental associations concerned, together with all other national organizations dealing with dental education or certification to send delegates to a meeting to be held in St. Louis on October 23rd to organize an Advisory Board for Dental Specialties. This Advisory Board should formulate standards and procedures for the organization of Specialty Boards by the various specialty associations. It is hoped that by proceeding in this manner a coördinated step may be taken which will avoid the uneven efforts of the different specialties in organizing boards without reference to the development of dental specialties and advanced dental education as a whole.

In proceeding in this manner Dentistry is using the experience which Medicine has gained over the last twenty years. After some years of unsatisfactory experience with boards established by the specialties in Medicine with their varying purposes, standards, and procedures, an Advisory Board for Medical Specialties has been organized which is composed of delegates from each of the specialty boards as well as each of the national medical educational and certifying bodies. This advisory board has established standards of preparation for specialists, procedures for organizing and conducting specialty boards and for publishing the names of qualified specialists. In doing so it has elevated the whole field of graduate medical education and put the preparation and certification of specialists on a sound foundation.

As an outcome of the organization of dental specialty boards with their requirements for advanced education and training on the part of candidates for examination and certification, it may be expected that graduate education in dentistry will receive a great impetus. While there can be no justification for so prolonged a period of university graduate work and hospital training in preparation for dental specialization as that proposed for medical specialists, still the preparation should be thorough and adequate. A two academic year course leading to a master of science degree plus some years of dental practice would seem none too much. Periodontology is very much more than a technic that can be gained in a few weeks of post graduate training. A two year course may seem excessive on first thought but, mature consideration of all the advanced subject matter in pathology, anatomy, bacteriology, biochemistry, nutrition, endocrinology, dermatology and medicine that should be included besides the theory and practice of periodontology, diagnosis, treatment planning and mouth rehabilitation will indicate that two academic years is a minimum.

In the case of older periodontists, who at the time of the organization of the board, are well recognized by the profession as qualified in the specialty, the examination and certification would necessarily be less rigorous in its insistence on science and theoretical learning. Some practical method can surely be established by which well recognized periodontists can be fairly examined and certified without opening the gates to all those who claim to be specialists at present but are really not highly qualified.

A matter which has confused the thinking regarding the certification of specialists, is the distinction between one qualified in a specialty and one limiting his practice to a specialty. While it is desirable that many should become qualified and certified as periodontists it is not evident that many should limit their practices to periodontal technics apart from the mechanical reconstruction of the mouth. It may be in the public interest in orthodontia or oral surgery for the specialty boards to insist that those certified shall limit their practices to their specialty but this should be left to the discretion of the boards concerned and not made universal. In periodontology the mouth rehabilitation is an essential part of periodontal treatment and cannot well be separated from it.

The establishment of specialty boards with the accompanying development of graduate education in dentistry should certainly do much to increase the number of highly educated and trained practitioners capable of leading the profession on to new and higher attainments in learning and service.

—H. J. L.

Certification of Periodontists*

By EDWARD B. SPALDING, D. D. S., F. A. C. D.

In 1914 the dental profession had no recognized specialty except Orthodontia. Today it has a number of accredited and self-appointed specialties. In 1914 there were few specific sections in the American Dental Association, or the National Dental Association as it was then called, and there was little interest in matters pertaining to periodontia. It was not until 1922 that an effort was made to establish a section in Periodontia—Periodontia being a term originated and adopted by the Academy. Officers of the National Dental Association came to the Academy and asked it to give up its organization and merge itself into a section of the larger society. The most of us know what the reaction to that request was by this group of earnest workers who had labored hard for eight years to improve themselves in their special field and to bring the Academy to a state of recognition. We are here today as evidence that part of that request was not acceded to, but we are also here to attest the fact that the Section of Periodontia in the American Dental Association is one of the best conducted and best attended in that organization and has been sponsored and guided since its inception by members of this Academy. The officers of that section have always held to the ideals maintained in the Academy and have provided programs that have been outstanding, of both scientific interest and practical value. This has generated an increasing interest in periodontia by the profession generally.

The American Academy of Periodontology has never closed the doors of its meetings to any one interested in periodontia and has always invited all members of the American Dental Association to attend. These things are mentioned to show that the Academy has always stood for service, in the best interpretation of the word, to dentistry and to the public, and has not side-stepped its responsibilities. It was founded and exists with one chief purpose and that, the study for the conservation of the supporting structures of the teeth. Without a foundation there can be no superstructure. Without a periodontium there can exist no teeth. Without teeth there can be no dentistry, only monuments, crutches and wooden legs.

The principles which underlie periodontia should penetrate, permeate and guide all other branches of dentistry, and yet when the periodontist sees operations, restorations and substitutions placed in the mouth without consideration for their effect upon the supporting structures of the remaining teeth, he wonders—wonders if the emphasis placed upon the technic of building upon the superstructure of the individual tooth may not blind the dentist to the fact that a tooth is no better than its foundation. He wonders, too, when we boast about dentistry being a scientific profession, if sometimes we forget that service is the object of its existence and that the public will think little of our boast unless the results of research and discoveries of science are made possible to the patient through the individual dentist's personal skill.

The effort to slip from under responsibility—to "let George or Mary do it"—under the claim and delusion that more are being served, does not enhance the scientific standing of Dentistry, nor its reputation for professional service.

We have observed that the periodontist wonders, but the general practitioner also wonders. He questions if this self-appointed specialist, the periodontist, has a right to his claim that "pyorrhea can be cured," and if so, where did he get his superior knowledge and skill?

Let us consider the definition for a specialist. The best definition we have found is the one given by Dr. J. Ben Robinson. It is: "A specialist is one who, in fact, is superior in a well defined department of work and whose superior status is established by study, training, and experience." Should we wonder that the general practitioner looks askance if some one of his profession claims more skill in a certain field than he?

Furthermore, the public is in a quandary. The patient no longer expects one small

*Presidential Address. Read before the American Academy of Periodontology, July 8, 1937, at Atlantic City, N. J.

cranium to know all and be expert in every branch of dentistry. He has learned this in obvious ways; but when he needs services of a particular nature how is he to determine who can give him this expert service? Unless he has honest advice from a general practitioner who knows the specialist intimately enough to be able to judge his work, the patient has no means of determining whether a certain self-appointed specialist has made honest study, received training and had the experience which fits him to deserve his claim. Often the patient learns to his regret that the specialist's claim was beyond his ability. He may also find that the work is delegated to assistants, not dentists, whose meager training does not fit them to grasp the seriousness of the work nor supply the knowledge or ability to cope with it. The public is beginning to ask questions. Some of the public may be fooled for a long time, but all the public is not going to be fooled all of the time. Does not the public need some means of protection?

Does not the general practitioner of dentistry deserve some means by which he can protect his patients and practice from the unfitted and unscrupulous self-appointed specialists in all fields of dentistry? Lastly, does not the accredited specialist himself need protection from those who have no right in his field as competitors? Is his study, training and experience gained at great sacrifice to be valued so lightly that he has to defend himself against the unqualified up-start?

These questions, ladies and gentlemen, will have to be answered, but the solution will not come this year, nor next, for they will require much thought, study, and careful decision. There will be a solution however, for those with vision have seen the handwriting on the wall and are taking steps to work out some solution which will be just and beneficial to the public, to the general practitioner, and to the specialist.

By referring to the Preamble of the Academy's Constitution we find two terms mentioned, "specialist" and "exclusive specialty" as being a necessity to the highest skill in periodontia.

Some six years ago the National Association of Dental Examiners began its study of Dental Specialization and the Certification of Specialists. For at least two years the American College of Dentists has had a committee studying the subject. To show that action is being taken to recognize, define and certify specialists, the state of Illinois has recently passed a law defining five specialties in Dentistry viz., that of Exodontia, Orthodontia, Prosthodontia, Periodontia, and Pediadontia, and has provided rules and means for examining and licensing applicants. The states of Tennessee, Oklahoma and Arizona, each have an Orthodontic specialist's law, and other states will probably follow. There are twelve specialties in medicine whose qualified members receive certificates of recognition, and their members are enthusiastic about the benefits of certification. The Committee on Dental Specialists of the National Association of Dental Examiners, the chairman of which is W. F. Bell, a member of this Academy, last year in San Francisco presented a very lengthy report and to show the seriousness in which they are dealing with the subject, we will quote from the beginning of the report: "This committee, in its endeavor to make an analysis of Dental Specialization, has interpreted its function to be the determination of the present status of Dental Specialties, the educational facilities for post-graduate training of the specialist, the possibility of legislative control and the advisability of recommending changes in the present set-up. With these objectives in view, it was decided that the institutions of dental education, the State Boards of Dental Examiners, the general practitioners, and the specialists themselves, could perhaps give valuable information by an expression of their reaction to the present system. For this purpose a questionnaire was sent to the Deans of the various Dental Colleges, the Secretaries of the State Boards of Dental Examiners, to thirty general practitioners and to thirty specialists throughout the country."

To state briefly the results of the questionnaires, they revealed that all felt that the present system of specialization is inadequate—"that the public is not sufficiently protected, and that better train-

ing and certification are necessary for this purpose."

Dr. Bell feels that the problem will be solved by the united effort of the specializing groups and the educational institutions, and that the first step is the establishment of sources for graduate instruction. When these have been established then the profession can demand, by suitable means, that the specialist equip himself to properly serve the public. Dr. Bell has appealed to the American Academy of Periodontology to help work out the problem of the specialist in periodontia.

Is it not logical that this oldest, largest and most consistant group of workers in periodontia should be the one expected to give the needed help? If the members of the Academy feel that this rquest is timely and worthy of support, your President feels sure they will accept the responsibility. He, therefore, recommends and urges that at this meeting now in session, a special committee of five be appointed to work upon specialization and certification as it applies to periodontia. He further recommends that this committee of five shall include the individual members of the now existing Educational Planning Committee on account of the importance of their previous work on problems which will confront the new committee on specialization and certification. This does not mean the abandoning of the old committee nor any of its planned work, only the augmenting of a new committee. While most committees of three work best, your President feels that the subject of specialization and certification will comprise so many angles of the practice of dentistry that the larger committee may be necessary for their proper consideration. Then, too, a questionnaire sent to every member of the Academy will probably be necessary, but, of course, that will be decided by the committee.

This is a problem which may take several years to work out satisfactorily, therefore, the selection of the two other members for the committee must be done with due consideration for the work, and your President recommends that they be chosen by the Council in Special Session for that purpose.

The suggestion for the certification of specialists in periodontia may give rise to a question in the minds of some of our members as to whether it will be necessary for all Academy members to become certified. Dr. Bell is of the opinion that at first it would probably be a voluntary procedure which would naturally not affect membership in the Academy. Later developments may however change this conclusion.

The Academy, represents a body of sincere workers in dentistry, but to be a scientist in the strict sense of the word is given only to the few. We all can and should, however, support as far as we are able every movement to advance the scientific aspects of dentistry.

Dentistry has definitely advanced along scientific lines in recent years due largely to the efforts of a man who with unusual vision saw the latent possibilities of dentistry in the field of science. This foresight of Dr. William J. Gies materialized first in the founding and publishing of the Journal of Dental Research, next in the organization of the International Association of Dental Research, and more recently, as a result of his efforts, the recognition of dentistry by the American Association for the Advancement of Science.

As members of an Academy we should at least be familiar with the advancement of dental science which is being regularly recorded in the Journal of Dental Research and our continuous support of this publication through one of the many forms of subscriptions is therefore strongly recommended for your serious consideration.

In closing, your President wishes to leave two suggestions for your consideration, one, that the certification of specialists is an important problem which the Academy should aid in solving as it pertains to periodontia, and the other that the Journal of Dental Research is worthy of the support of every member of this organization because of its basic value to scientific research and to the progress of dentistry.

The Quarter Century Mark

Deep-rooted in sincerity and strengthened by the experience of mistakes, the American Academy of Periodontology has grown steadfastly for twenty-five years and stands today indispensible in the Profession of Dentistry.

Probably no other group in any profession has built more strongly upon the personal ties and friendships of its members. A spirit of harmony, entirely apart from political or partisan feelings, has nourished the Academy through all of these years. No member has yet been given cause to criticize its aims, its ambitions, or to regret his association. Honor has been bestowed on everyone privileged to take part in its development.

In October of this year members will have their first opportunity to pay homage to their Academy and to the courage and foresight of its founders. Every member should esteem it an obligation to personally present his felicitations to the Academy on this occasion. The program, entertainment, and special social activities of our annual meeting will revolve about this anniversary.

Members of the American Dental Association are again most cordially invited to attend our sessions. However this year we may be pardoned for thinking more of our own privileges and pleasures. This is to be above all a meeting for members, conceived and planned in their pride of possession. Here will be an opportunity to honor one another, to appraise achievement, and above all to gather enthusiasm and ambition for a greater future for the Academy.

Plan to attend the Silver Anniversary of your Academy.

CLAYTON H. GRACEY, *President*

Resumé of Report of Pathologic Conditions Associated with the Use of Sodium Perborate, Observed by Members of the American Academy of Periodontology*

IN RECENT years, the promiscuous and unsupervised use of sodium perborate as a powder, in solution, as part of a dentifrice or added to the dentifrice by the patient at each use, has become a part of the routine of a large portion of the lay public. The following conditions have been recorded as associated with, and evidently caused by, such use of the chemical:—

Item 1—painful, extensive chemical burns of the oral mucosa (including the gingivae);

Item 2—painless chemical burns producing a milky-white discoloration, especially of the marginal gingivae;

Item 3—edematous condition of the oral mucosa generally, causing it to become tender or predisposing it to ready abrasion and infection through minimal traumatization;

Item 4—discolored patches of hypertrophied papillae of the tongue producing a form of "hairy tongue" which in some instances causes gagging, or irritation of the soft palate or pharynx;

Item 5—cervical hypersensitiveness.

QUESTIONNAIRE

Approximately how many of the respective items mentioned in the accompanying letter have you had occasion to observe? Please signify by underlining the words.

ITEM 1—

Perborate used in the form of:

None
Few (mouthwash—tooth powder—
(part of dentifrice—added to
toothpaste
Many (mouthwash—tooth powder—
(part of dentifrice—added to
toothpaste

*This report is based on answers to a questionnaire sent by I. Hirschfeld, Chairman of the Committee on Scientific Investigation, June 12, 1937. Read before the American Academy of Periodontology, July, 1937 at Atlantic City, N. J.

ITEM 2—
None
Few (mouthwash—tooth powder—(part of dentifrice—added to toothpaste
Many (mouthwash—tooth powder—(part of dentifrice—added to toothpaste

ITEM 3—
None
Few (mouthwash—tooth powder—(part of dentifrice—added to toothpaste
Many (mouthwash—tooth powder—(part of dentifrice—added to toothpaste

ITEM 4—
None
Few (mouthwash—tooth powder—(part of dentifrice—added to toothpaste
Many (mouthwash—tooth powder—(part of dentifrice—added to toothpaste

ITEM 5—
None
Few (mouthwash—tooth powder—(part of dentifrice—added to toothpaste
Many (mouthwash—tooth powder—(part of dentifrice—added to toothpaste

Additional items observed by you and form of sodium perborate used therein:—

Tabulated Summary of the Replies to the Questionnaire

Questionnaires sent out to members of the Academy	140
Filled out questionnaires returned to the Committee	90
Members claimed (by themselves) as unqualified for report, because of limited experience with sodium perborate	3
No replies received from	58
Lesions observed by	82
Lesions observed, but no detailed information given by	2
Lesions observed, and detailed information given by	80

Among the 80 questionnaires giving detailed information, the incidence of the respective pathologic Items enumerated in the questionnaire was as follows:—

	"few"	*"many"*
Item 1	36	15
Item 2	39	9
Item 3	30	17
Item 4	34	10
Item 5	22	19

Thus it will be observed that the total number of questionnaires that indicated positive findings were distributed as follows:

Item 1	51
Item 2	48
Item 3	47
Item 4	44
Item 5	41

The lesions were associated with, and presumably caused by, sodium perborate used in the following manner:

	Item 1	*Item 2*	*Item 3*	*Item 4*	*Item 5*	*T.*
(a) As a mouthwash	17	14	20	13	11	75
(b) As a tooth powder	20	16	24	13	17	90
(c) As part of a dentifrice	7	9	4	1	9	30
(d) Added to toothpaste	3	1	0	0	4	8
(e) Not specified	17	21	16	12	10	76

Lack of space does not permit enumeration here of various significant individual remarks contained in many of the returned questionnaires. (These will be published elsewhere.) However, it seems pertinent to call attention at this time to one noteworthy observation made by some: namely, that, according to clinical observation, it has yet to be proven that the daily use of sodium perborate in any form is of any particular value in the prevention of Vincent's infection or its recurrence.

The committee is thankful for the fine coöperation of the members of the Academy in filling out the questionnaires and for the valuable information contained therein.

730 Fifth Avenue
New York City.

Case Reports

Apparent Selection of Similar Areas Other Than the Gingival Crevice by Spirochetes and Fusiform Bacilli in Five Related Cases of Infection.

By BENJAMIN TISHLER, D. M. D.
Boston, Massachusetts

The great majority of Vincent's Infection cases that we treat are isolated ones. Occasionally a husband who has become infected will refer his wife for an oral examination, or the case may be reversed, or one friend may refer another and it is found that one has contracted the disease from the other. The lesions appear on the marginal gingiva in most instances although there may be additional areas affected in any part of the oral cavity, as we all know. It was my privilege to treat five interesting cases of this disease at two different periods, the details of which follow:

The cases, two in one group and three in another were as follows and will be described in the order which they were seen. Carl A., a senior student at Harvard, 21 years of age, was referred by his dental practitioner who had unsuccessfully tried to treat a severe case of oral ulceration for a week when the first symptoms were noted. An examination disclosed a marked lesion of the right and left mucous membrane involving the junction of this alveolar and areolar mucous membrane of the entire maxilla. There were areas on the membrane of both cheeks and hard palate, also beneath the tongue, on the orifices and over both submaxillary ducts but none on the marginal gingivae anywhere. Both submaxillary glands were tender to touch, the breath was foul, tongue coated and saliva ropy. Patient reported that he could not eat solid foods in comfort so his diet was confined to milk and eggs. He was a heavy cigarette smoker. A smear showed a multiplicity of spirochetes and fusiform bacilli.

The following day, his roommate, J. H. C., 21 years of age, asked for an examination saying that he had inadvertently used his friend's tooth brush a few days previously. An examination disclosed typical ulcerated areas with a red outline on the buccal gingiva in the region of the right inferior second molar and on the right buccal mucous membrane. There were others on the labial mucous membrane in the left superior lateral and cuspid area, also at the junction of the maxillary areolar tissue and buccal mucous membrane and under the tongue. The lesions were not as marked as in the case of his roommate but the striking similarity was the ulceration at the junction of the maxillary areolar and buccal mucous membrane in both of these cases and the fact that the marginal gingivae were not ulcerated in any part of his mouth. No smear was taken in this case because the etiology and character of the lesions were so apparent.

The second group of three cases was as follows: A. M., male, age 35, was referred by a friend for treatment. He had been confined to his bed for several days with a case of influenza but his oral condition was so painful that he wanted it relieved. The fact that he left his bed to come to my office was not known to me when he telephoned for an appointment, otherwise, I would have called on him at his residence.

His complaint was that his gums were very tender to touch and bled when he brushed his teeth. The first symptoms were noted four days previously. His throat was sore and it was difficult for him to swallow food of any kind. An examination disclosed sloughing on the areolar tissue in the right inferior buccal region and another area about 4 mm. long and 1 mm. in width on the left labial and a smaller one on the left buccal mucous membrane; the labial areolar tissue beneath the lower anterior teeth was badly sloughed also. Temperature 100.6°, appetite poor, saliva stringy and tongue heavily coated. Smear examination showed a multiplicity of spirochetes and fusiform bacilli. No ulceration on the marginal gingivae was noted anywhere.

Miss L. B., age 22, referred by the preceding patient, was seen about a month later; reported that her mouth became sore

about a week and a half previously. Lesions similar to his, i. e., on the buccal and areolar mucous membrane were noted. This patient developed many other lesions which involved the areolar, labial and buccal mucous membrane, mucous membrane under the tongue and some as far back as the internal border of the left ramus; others were noted on the mucous membrane over the sub-maxillary glands. She had a neurosis which caused her to bite her cheeks and as a consequence, these areas were ulcerated. The marginal gingivae were unaffected anywhere but more lesions developed than in any of the other four cases reported in this paper.

Miss D. C., age 25, was referred for treatment by the patient whose case has just been described, twelve days after the former's first visit. This patient reported that she noted her mouth became sore two days previously in an area on the right side of the palate in the molar region. Another developed on the lip and on the labial mucous membrane in the region of the right inferior cuspid. It was about 3 mm. long and 4 mm. wide. There were other areas at the junction of the alveolar and areolar mucous membrane, over the right superior lateral and at the junction of the left labial and alveolar mandibular mucous membrane, and in the region of the left superior lateral. The mouth of this patient was the least affected of the entire group. A significant fact regarding these three cases was that they ate all their meals in the same restaurant. The lesions in all five cases healed after being treated by the usual medicaments employed in Vincent's Infection cases.

The interesting characteristics of the two groups of cases were:

(1) There was apparently an etiological relationship between them.

(2) There was a preponderance of spirochetes and fusiform bacilli in the examined smears.

(3) There was marked ulceration present.

(4) All cases responded to the usual Vincent's Infection treatment.

(5) No lesions were found on the marginal gingiva anywhere.

356 Commonwealth Ave.

Stomatitis—Caused by Chemical Burns Due to the Use of Pure Sodium Perborate*

By MARTHA E. JONES, D. D. S., Detroit, Michigan

The patient, a young woman, was brought to my office by her physician, who questioned whether she presented a case of Vincent's Infection.

On examination, I found ulcers on her swollen lips, gingivae, the buccal mucosa, and fauces; her tongue and throat were so swollen, that she could swallow only with the greatest difficulty. She was pallid and, at the same time, feverish. She had lost 10 pounds in weight. She did not, however, carry the distinctive foetor peculiar to Vincent's. Incidentally, she was suffering from a severe case of vaginitis.

The oral condition did not, in any way, present an appearance of any case of Vincent's I had ever seen, and, with the usual stench of this disease being absent, I suggested to her physician the routine laboratory test for Vincent's—to which she was negative.

The following smears were negative also: *Gonococci; Diphtheria Bacilli*—but cultures showed staphylococci and streptococci; *Treponema Pallidum; Trichomonas Vaginalis*—but smears showed numerous pus cells and secondary organisms.

The case was complicated by an extreme Class II malocclusion, produced many years previous by the removal of two lower premolars, in an attempt to correct her occlusion, so, the patient bit with her lower incisors, directly into the palatal rugae, where most of the soreness appeared. Hence, in swallowing, when she closed her jaws, she continuously aggravated this swelling, posterior to her upper incisors.

Lest you question why I did not do something to correct this condition, let me say, that she was a most uncoöperative patient. Grinding the incisors would give her no permanent relief, and she would not wear any kind of a splint. So, at some future time, upon bruising the palate, the

*Read before the American Academy of Periodontology, July 9, 1937, at Atlantic City, N. J.

patient may look for inflammation, though not to the extent of the injury to the tissues produced by the sodium perborate.

Before I outline the treatment eventually evolved, I wish to tell you of two preparations I used, hereafter referred to: The first of these is Caroid; the second is Resorcitannol. The efficacy of Caroid was brought to my attention several years ago by a physician in Detroit, formerly of the Staff of Henry Ford Hospital.

1. Caroid is a vegetable ferment, it is not disagreeable to the taste, and it has the virtue of dissolving dead tissue and mucous. It does not liberate gas.

Where the mucous membranes are inflamed, so that the patient is in almost unbearable pain, and the area is nearly inaccessible—such as fracture cases, complicated with Vincent's Infection, I use a 15% aqueous mixture of Caroid to bathe the tissues, prior to medication. Where there is a throat complication, such as in the case under discussion, the patient may swallow Caroid, with excellent results in cleansing the mucous membranes of the throat.

Caroid has another virtue—it is distinctly soothing. In brief, it cleanses the mucous surfaces, preparing the way for efficient medication.

2. Resorcitannol: When, at last, we found the cause of her stomatitis and vaginitis—chemical burns, caused by sodium perborate, Dr. Frank W. Hartman, head of the Department of Pathology at Henry Ford Hospital, prepared for me a solution, which he calls Resorcitannol. It is a 5% solution of tannic acid in hexylresorcinol. It is highly astringent, soothing, and healing.

Treatment

Initial Treatment—Before tests were made—Mapharsen by Parke, Davis & Company—used dry from the ampoules, delicately, with wood points.

Following two days—Caroid, used as a swab several times a day, but particularly before medication by Abbott's Potassium bismuth tartrate-Metoso—as the mouth wash. It is interesting to note that at the end of the third treatment the patient could swallow with some degree of comfort.

As has been stated above, she was not a coöperative patient, so, at the end of the third day, we placed her in a hospital, in order to have our instructions carried out.

Fourth Treatment—Saturated solution of copper sulphate to try to reduce the swelling, and lessen soreness, posterior to the incisal region. The patient objected vigorously—and effectively—to a second application of copper sulphate. In any case, it accomplished nothing.

It was at this stage, after wandering about in a wilderness, so to speak, that we learned that she had been brushing her mouth with pure sodium perborate—for many weeks—on the recommendation of her dentist, though she had no history of Vincent's Infection. It is significant to note here also that at this time, her physician learned that she was using a vaginal douche, which he found contained a high percentage of sodium perborate.

From this time on, the treatments were simple, definite and effective. Ten consecutive daily treatments. Her mouth was swabbed four times daily, after meals, and before retiring—with Caroid. Mouth wash—normal saline solution. Resorcitannol—for night treatment. Inflammation markedly reduced.

At the end of 14 days, she had so improved, with the nourishment she had been able to take, that she was strong enough to present herself at my office. I polished her teeth thoroughly. The area posterior to the upper incisors, was still puffy, so I touched it with pure nitric acid followed immediately with a spray of water. Other areas treated with Abbott's Potassium bismuth tartrate.

At intervals of two days, for two treatments, I repeated the polishings, for she was not able to brush thoroughly, and followed the polishings with Potassium bismuth tartrate as a swab. Home treatment, normal saline solution as a mouth wash. The tissues of her mouth were normal at the end of three weeks.

525 David Whitney Bldg.

Endocrine Therapy In Atrophic Periodontoclasia

By WILLA YERETSKY, D. D. S., Grand Rapids, Michigan

Periodontists have for many years recognized a type of periodontoclasia which was apparently due to some obscure cause rather than a local one and which was disappointingly resistant to treatment. This type was said by Willman to occur in about five per cent of cases. It has been known as atrophic dystrophic periodontoclasia.

Various theories have been advanced as a possible etiology. In general it has been recognized as systemic. Some investigators have believed the cause to be nutritional.

More recently Becks advanced the theory that the etiology was of endocrine origin but has made no published statements as to which group of endocrines he considers responsible.

It is known that the thyroid gland has a very important function in the calcification of teeth during their formation period. The parathyroid governs calcium metabolism throughout life. Hence, it would be logical to suppose that this group might have an important part to play in the cause of dystrophic periodontoclasia.

According to Marinus, a leading endocrinologist of this country, administration of thyroid or parathyroid extracts after eruption of the teeth does not influence calcification of the teeth and neither does it influence the osteoparosis and halisteresis of dystrophic periodontoclasia.

Marinus states positively that these cases are due to a hypo-gonadism. They are most apparent in women and can be aided by the administration of whole ovary and such other gonad hormones as may be indicated in the individual.

The author has had a case of dystrophic periodontoclasia due to hypo-gonadism under observation for two and a half years. This patient reported for treatment in October, 1934. She was then twenty-nine years old, married and had one child aged two. The alveolar margins showed extensive resorption for so young a patient. There were irregular deep pockets, a profuse discharge of pus and ready hemorrhage from the gingivae. Many of the teeth were mobile and the patient was very apprehensive of losing her teeth. She was of an intelligent class, well-nourished and consumed an adequate well-balanced diet. Her appearance led one to suspect some endocrine disturbance since she had a hypertrichosis of the face, and cried readily at any slight disturbance. Her basal metabolic rate was within normal limits. Upon consultation with her physician it was decided to refer her to Marinus in Detroit, who placed her upon whole ovary therapy which has been continued. During this period she has had one pregnancy with a normal delivery and a normal child. Her oral condition has continued excellent. She has no further secretion of pus and has very little deposit of calculus between prophylactic visits. There is marked regeneration of bone visible in the X-ray, which I shall show you.

It may well be that the field of endocrinology will furnish the solution of that percentage of cases whose cure for so many years has baffled us.

26 Sheldon Avenue.

Localized Hypertrophic Gingivitis Due to Tongue Habit

By JAMES E. AIGUIER, PH. G., D. D. S., Philadelphia, Penna.

Patient, female, forty-six years of age, nervous type, referred for treatment. Examination showed the two maxillary first incisors, vital teeth with porcelain jacket crowns, which had been on for six years.

There was a marked hypertrophic gingivitis associated with the four maxillary incisors, more pronounced over the first incisors. This condition had existed for some months, the patient having been to several dentists, but receiving no relief.

The gingiva showed considerable hypertrophy, bulging labially from the interproximal spaces and around the cervex; it was purplish in color. The occlusion showed some trauma. Smears revealed a mixed infection with some Vincent's organisms. Gingiva was treated with vari-

ous medicaments to eliminate these organisms, using solution chromic acid 8%, acri-violet 5% and dilute Talbot's solution of iodine. Occlusal trauma was relieved, the calculus and debris removed and tissues gently massaged.

The patient was instructed in mouth care, selection and proper use of a suitable toothbrush. Massage was suggested, using gauze on finger with application of soap for a lubricant. She was advised to hold warm saline solution (½ teaspoonful of salt to full glass of warm water) in mouth every two or three hours, each mouthful to remain in the mouth one half minute. In three weeks time there was a marked improvement, but the condition did not disappear.

As there was some question regarding the crowns playing a part in the condition and since there was a marked gingival recession, it was deemed advisable to remove them. After some additional preparation of the roots, new crowns were made and placed.

The patient left for a vacation of three months. When she reported we found the hypertrophied condition was the same as when she was first referred to us. In studying the patient we noticed from time to time, the tongue would find its way up to these teeth. Upon calling the patient's attention to this, she acknowledged that she had developed a habit of pressing her tongue against the teeth.

Searching for a cause, we found the jacket crowns were somewhat bulky on the palatal side which apparently annoyed the tongue and this developed the pernicious habit, the pressure exerted causing the disturbance.

With stones and disks the crowns were ground down and polished, patient cautioned about checking tongue habit. There was evidence of improvement in a few days and eventually this condition disappeared.

Medical Arts Bldg.

"Doctor, What Dentifrice Do You Recommend?"

Among the bromidic questions asked by dental patients, "Doctor, what dentifrice do you recommend?" is heard almost as frequently as, "Doctor, will it hurt?" or "Doctor, are you going to use the drill?" Have we a satisfying answer to this question regarding dentifrices which is in keeping with the importance of the subject to the patient? The common reply "Anything will do," will no longer do. The radio ballyhoo and false claims through other forms of advertising have aroused suspicion among intelligent people. Dental patients expect, and rightly, truthful and accurate advice on this subject from their dentist which is implied in the form of address—"Doctor."

Unless a dentifrice is accepted by the Council of Dental Therapeutics we had better be cautious about advising its use unless we are familiar with the formula and its effect upon the teeth and gingiva. It is especially desirable that we know its abrasive action.

There are dentifrices on the market which are positively harmful to the teeth. Others are injurious to the gingiva. There are those over which there is no satisfactory control of formula since it may be changed at any time for trivial or financial reasons to suit the whims of those who are selling it.

To meet this situation some of us have advised the use of Precipitated Calcium Carbonate as an economical dentifrice and as we thought a safe one. That there are various physical properties such as particle size and abrasiveness should be noted and if this material is recommended we should specify these factors to the druggist when we prescribe it. Some have approved the use of equal parts of sodium chloride, bicarbonate of soda, and sodium perborate. The fineness of sodium chloride is most important and when it is used in a dentifrice should be very finely milled. As to sodium perborate in a dentifrice, the blanket of suspicion rests upon it at the present time. In any case if used in the mouth it should be for a specific purpose and with definite limitations, as should all drugs.

A recent edition of *Accepted Dental Remedies* devotes many pages to dentifrices and their various ingredients. We can, to advantage, refresh our memories on this subject and add considerable to our present knowledge in order to be able to protect our patients from exploitation.

BOOK REVIEWS

The Avitaminoses by Walter H. Eddy, PhD. and Gilbert Dalldorf M. D. 338 pages. 1937. The Williams and Wilkins Company.

The authors of 'The Avitaminoses' who have themselves contributed many researches in this field, have rendered a most valuable service in collating and digesting the enormous literature about vitamins. They have presented it in form so that practical use can be made of it by physicians and dentists in detecting and treating disturbances caused by slight deficiencies.

In part I of the book they have taken up each vitamin as to its nature, function, and the clinical, subclinical and anatomical manifestations of deficiencies. The detailed pathology of the various tissues caused by partial and extreme deficiencies are especially valuable contributions. This is followed by chapters dealing with the vitamins and resistance to infection, effects of dietary experiments including those on dental caries susceptibility, and the vitamins and blood regeneration . . . In part II they take up the methods of assaying for vitamins, the clinical tests for vitamin deficiencies and tables of vitamin values of foods. A good author and subject index completes the volume.

Among the mass of interesting information are many facts of great interest to those dealing with dental problems. Perhaps the most important is the fact that emerges after careful reading of the whole book, that human deficiency disease is seldom limited to one deficiency. The various protective vitamin and mineral elements support each other so that a deficiency in one causes a relative deficiency in others that otherwise would not be deficient. Also diets ill balanced in proportions of protein, carbohydrates and fat, especially if high in carbohydrate, require greater amounts of some of the vitamins than normally are required. Infections and toxins also cause destruction of some of the vitamins and thus the vitamin requirement is higher where infection is present. Gastro intestinal disturbance which may be caused by several of the single deficiencies adds to the deficiency effects by failure to absorb the vitamins ingested. The vitamins are intimately related to the endocrine glands and hormones; for instance hyperthyroid intoxication tends to be neutralized by a sufficiency of vitamin C.

All of these facts indicate the need of optimal amounts of the vitamins and in some cases more than the usual optimal requirements. Evidence is advanced to show that any of the five vitamins which are known to be necessary for man may easily be deficient in the diet and in fact are often deficient.

Vitamin A, the growth and anti-xerophthalmic vitamin, is found most abundantly in haliver and cod liver oil, butter, eggs, animal liver and many vegetables and fruits. Many diets are not well supplied with these foods and are deficient in this vitamin. A new test for a slight deficiency is a test for partial night blindness. Application of this test followed by vitamin A administration to recovery indicates that à large percentage of persons, especially children, are deficient in this vitamin. This fact is particularly important in dentistry because deficiency in the vitamin lowers general resistance to bacterial invasion and this probably contributes to gingivitis and periodontoclasia.

Vitamin B or B1 is the antineuritic or anti beri-beri vitamin. It is found most abundantly in the germs of seeds, the coatings of grains, in yeast and in many vegetables and fruits. In mild deficiencies the first effects are on the alimentary canal. Evidence is advanced to show that a great proportion of the digestive disturbances of man are due to deficiency in this vitamin. Deficiency in vitamin B also leads to lowered resistance to bacterial invasion and may thus contribute to periodontal disease although no evidence is given that gingivitis or periodontoclasia are directly caused by such deficiency. A possible relation of vitamin B deficiency to diabetes is suggested by the fact that some mild cases of diabetes have been benefited by administration of dried yeast to the

point where insulin was no longer necessary. No specific test has so far been devised to determine slight deficiency of vitamin B. The benefits that come to many in the form of increased appetite, freedom from constipation and other digestive complaints by the administration of yeast or wheat germ indicate that mild deficiency of vitamin B is fairly common.

Vitamin G the antipellagra vitamin is discussed in a chapter called "The Resolution of the Vitamin B Complex." In this chapter a number of factors which have been obtained from wheat germ and yeast and found necessary for the nutritive requirements of certain experimental animals, are discussed. The relation of deficiency of these vitamins to pellagra, sprue, pernicious anemia, spinal cord sclerosis and coeliac disease, is taken up. The subject is in a very confusing and contradictory state as yet but the evidence indicates the extreme importance for mankind of unraveling it.

Knowledge about vitamin C, the antiscorbutic vitamin, is in a better state since its chemistry and tests for subclinical deficiencies are well worked out. The first effect of deficiency is capillary fragility. However, vitamin C deficiency is not the only cause of capillary fragility. The second effect is on the tooth pulp and then the periodontium. The effect is apparently on the intercellular material of connective tissue. The authors give evidence that gingivitis in children is very largely a vitamin C deficiency condition. Vitamin C is rapidly destroyed in infectious disease and is apparently much concerned in the natural immunity of the body to infection and toxins.

There are apparently several forms of vitamin D, the form found in cod liver oil not being that found from irradiated vegetable products. The authors believe the two forms are equally efficacious in treating and preventing human rickets and its adult counterpart osteomalacia. The relation between rickets and hypoplastic teeth is shown. The authors' evidence indicates that vitamin D deficiency is the most important single cause of dental caries susceptibility but they emphasize the point that a combination of factors undoubtedly play a part including adequate calcium, phosphate, vitamin A, vitamin C, increased bulk in diet, and decreased sugar. Overdosage with vitamin D is possible but only with a thousand or more times the ordinary therapeutic dose. None of the other vitamins appears to be toxic in any dosage.

The book is well written, is easy to read and is beautifully illustrated with photomicrographs and pictures of pathological specimens.

—*Harold J. Leonard, D. D. S.*

"In the present state of medical knowledge a pronouncement of the sentence 'incurable' on a patient places a serious responsibility on the physician and implies a greater knowledge than he possesses."

—DR. ERNEST P. BOAS.

PROGRAM

The Twenty-fifth Annual Meeting

AMERICAN ACADEMY OF PERIODONTOLOGY

St. Louis, Missouri

October 20, 21, 22, 1938

FIRST DAY: *Morning*

Reports of Committees

Presidential Address.....Clayton H. Gracey

Afternoon and Evening

The Historical Background of Periodontia

Contributions . . . by A. W. Bryan, I. Hirschfeld, Harold J. Leonard, J. O. McCall, Arthur H. Merritt, S. J. Parks, Willa Yeretsky.

SECOND DAY: *Morning*

Past-Presidents' Breakfast—Report.....Carlos H. Schott, Chairman

Future Trends in Periodontia.....Paul R. Stillman

Committee on Scientific Investigation—Report.....Edgar D. Coolidge, Chairman

What Happens to the Supporting Structure in Traumatic Occlusion?....Dr. Balint Orban

DISCUSSION LUNCHEON

Leaders:

R. Kronfeld L. Fonner

Afternoon

Clinics . . . by Hunter S. Allen, Lillian Barkann, T. E. Braly, Bernard Friedman, I. Hirschfeld, Carl W. Hoffer, Arvah S. Hopkins, J. A. Jacobsmeyer, A. F. James, James M. Prime, Carlos H. Schott.

FRIDAY EVENING

BANQUET

THIRD DAY

Radiodontic Service in Periodontal Practice.....Clarence O. Simpson

A New Treatment for the Interproximal Space and Septal Tissue.....James M. Prime

Election of Officers

4:00-6:00 Informal Tea

THE Journal of Periodontology is lovingly dedicated to the memory of Doctor Gillette Hayden. Her selfless devotion and untiring efforts in behalf of periodontia and the American Academy of Periodontology, have served as an inspiration to her close associates which can only be consummated by carrying onward the work for which she spent her life.

THE JOURNAL OF PERIODONTOLOGY

Published twice yearly—January and July—by

The AMERICAN ACADEMY OF PERIODONTOLOGY

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Publication and Editorial Office: 555 W. MAPLE AVE., BIRMINGHAM, MICHIGAN

Single copies of this number may be obtained from

Publication Office at $1.00 each

Subscription Price $2.00

All foreign subscriptions to be paid in United States money

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